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Scrapie and Related Myopathies in Sheep

Preliminary Observations on their Investigation and Attempted Control by a Voluntary Health Scheme

BY

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SCRAPIE has been the subject of discussion and controversy for a century and a half, and even to-day no generally acceptable definition of the disease, its clinical signs, pathological basis, and aetiology can be given. This is in part due to its very insidious onset, long and variable course, and the variety of clinical signs which can accompany the disease, and in part to the extreme reticence of flockmasters and shepherds to disclose whether they have the disease, so that our knowledge of its epidemiology is fragmentary. The principal features of the disease are a slow wasting of the body flesh, with varying degrees of unsteadiness of the gait and rubbing of buttocks, poll, and shoulders, and death after three to six months. At *post-mortem* examination no significant macroscopic lesions have been reported hitherto.

This paper reports the finding of a primary degeneration of skeletal muscles (myopathy) in sheep affected with scrapie. This myopathy has been found in all cases of scrapie examined, as well as in cases of several related syndromes occurring in scrapie-affected flocks, but in which rubbing is inconspicuous or absent. These syndromes, tentatively called inapparent myopathy (P1), posterior myopathic palsy (P2), post-partum myopathic pining (P3), acute myopathic asthenic paralysis (P4), juvenile myopathic pining (P5), and generalised myopathic ataxic pining (P6), have not, as far as can be ascertained, been described previously. In addition non-wasting forms of scrapie have been encountered. The evidence suggests that scrapie is but part of a more widespread myopathy, which gives rise to a number of clinical syndromes depending on the location, age of development, and extent of the myopathic process. We recognise nine different syndromes at present, in three of these rubbing is a conspicuous feature and the syndromes may be classified as scrapie, while in the other six rubbing is not conspicuous and these conditions would not be diagnosed as scrapie.

The aetiology of the myopathy is undetermined, but the balance of the evidence is against its being an infectious disease. It has many similarities to muscular dystrophy and dermatomyositis (polymyositis) in man. Most cases of the former disease are usually considered to be hereditary; the aetiology of dermatomyositis is obscure. Economically in flocks affected with myopathy the losses sustained from non-scrapie forms of the disease are often as serious as those from scrapie, and are thus equally important to the practical sheepman.

General Introduction

The disease is called scrapie, "rubbers," or "piners" in Britain, "Traubenkrankeit" in Germany, and "la tremblante" or "la maladie pruritique de mouton" in France. It has been disseminated widely in West European sheep stocks since at least the second half of the 18th century, when it was described in Germany and France in merinos and sheep of merino descent. The early accounts in Britain date from the 1790's and coincide with the importations of Spanish merinos under George III. The prevalence of the disease has fluctuated in different districts and in different breeds during the past century, although no reliable data on these matters can be obtained, but it is quite clear that from time to time the malady has been sufficiently serious to make flockmasters discontinue their breeding flocks, and this is the position in some areas of Britain to-day.

The cause of scrapie remains controversial. It was thought to be hereditary a century ago. M'Gowan

(1914) considered it was due to sarcocysts in the muscle, but this ubiquitous parasite can hardly be held responsible as M'Fadyean (1918) and Brownlee (1940) have pointed out. Stockman (1913, 1926), who wrote from a wide experience, thought it was more probably due to an infectious agent, a view to which M'Fadyean (1918) acceded. However, no microbe could be demonstrated and transmission experiments were unsuccessful. During the 1930's workers at Moredun (Greig, 1940) became impressed with the idea that it might be due to a virus infection with an unusually long incubation period. This suggestion was strengthened when scrapie developed in Blackface sheep, a breed which are said to have a very low incidence of the disease naturally, following the inoculation in 1935 of louping-ill vaccine prepared from the brains of sheep inoculated with louping-ill virus (Gordon, 1946). It was found that inadvertently the brains of several scrapie sheep had been used in the preparation of a certain batch of the vaccine, which contained 0.35 per cent. of formalin. About the same time, French workers (Cuillé & Chelle, 1936, 1938) reported the transmission of the disease by the intracranial inoculation of emulsions of the brains of scrapie sheep, and of Chamberland filtrates prepared therefrom, but the numbers of sheep used and the numbers becoming affected were too small to provide convincing evidence for their contention that a specific virus was responsible for the disease. Meanwhile, studies were continued at Moredun by Gordon (1946) and later by Wilson and his colleagues (Wilson, Anderson & Smith, 1950) in which they were able to set up the disease in serial passage, although the percentage of inoculated sheep which developed the disease was usually not more than 50 per cent. These results were reviewed by Wilson (1952) and were interpreted to mean that scrapie was due to a virus infection of the central nervous system with an incubation period of 18 months to five years. The virus could possibly remain viable on pasture for long periods (Greig, 1940). However, some of the experimental and much of the epidemiological evidence is difficult to reconcile with such a theory of causation, and none of the usual stigmata of virus infections is seen in microscopic preparations of the central nervous system.

A cardinal difficulty in studying scrapie has been the absence of any constant macroscopic or microscopic lesions which would account for the clinical signs of the disease. Besnoit & Morel (1898) thought they had discovered a degeneration of the peripheral nerves, which would have explained the wasting, rubbing, and ataxia, but their observations have not been confirmed and are generally thought to be due to artefacts. There are vacuoles in some neurones of the brain and spinal cord (Besnoit & Morel, 1898; Brownlee, 1940; Holman & Pattison, 1943), but they are variable and occur in clinically unaffected sheep, and in human diseases they are recognised as occurring in debilitating nervous diseases. Furthermore their location and number are such that it is difficult to accept that they can be responsible for the clinical signs. We are thus left with no satisfactory morphological basis for the disease.

In attempting to circumvent this difficulty various psychological factors have been postulated. It has been said (Greig, 1940) that the rubbing was due to pruritus and that the constant itching prevented the animal from resting and feeding properly, so that it lost weight, became weak, and wasted away. No factual evidence in support of this contention has been published and when one measures the individual food intakes of affected animals, one finds that their food consumption is more than adequate to maintain a healthy sheep in excellent bodily condition. We are therefore forced to seek some other explanation of the weakness and loss of flesh. The cause of the rubbing and of the development of the nibbling response when the sheep's back is rubbed with the fingers are also quite unknown.

During the past quarter of a century most investigators of scrapie have been preoccupied with the infectious nature of the disease. Virtually all the published work has been on the possible virological origin of the disease. The time is perhaps ripe to reconsider the pathological physiology of the disease, deciding in the first instance just what clinical signs may occur in scrapie and then attempting to provide some scientifically acceptable explanation for them.

Many of the clinical features of scrapie were well described by Stockman (1913) but when one has the opportunity of studying the disease as it occurs in many pedigree flocks one realises that there are many features of the disease which have not been generally recognised.

We therefore sought to define the clinical disease and have studied the clinical manifestations of scrapie and related diseases in about 450 animals, with the close co-operation of a large number of flockowners and shepherds in England and Scotland, who provided much detailed confidential information. An attempt to provide an explanation of at least the most prominent of these clinical signs led to an investigation of the skeletal muscles and to the discovery that these sheep were suffering from a severe myopathy. While several clinical features of the disease remain unexplained, the muscle disorder does explain a large number of the clinical findings. A preliminary account of these observations and of some of their practical implications is given below.

Material and Methods

Clinical cases

Of 448 affected sheep examined, 69 were studied closely, both clinically and pathologically, including a careful examination of the skeletal muscles (see Table I). These sheep came from 25 different flocks widely scattered over 12 counties in Britain. Some were from commercial flocks but the majority were drawn from closely shepherded pedigree flocks which are members of a voluntary Pedigree Sheep Health Scheme organised by the author. The members of this Scheme own about 5,000 breeding ewes and identify individually all animals from birth; they keep records of parentage, and of all ill-health, disability, and death. They are visited and their records checked at least twice a year. These flocks are self-contained, except for the introduction of new sires from time to

TABLE I

A PROVISIONAL CLASSIFICATION OF THE SYNDROMES OF MYOPATHY IN SHEEP

Type	Designation	Clinical signs	Outcome	Number of cases seen	Autopsy data		Microscopic findings
					Number	Principal lesions	
Group A							
1	Classical scrapie	Rubbing ; severe ataxia and weakness ; skin lesions	Fatal	316	36	Moderate wasting of skeletal muscles with pale " putty " foci (mainly class 1 lesions, but some class 2)	Severe and widespread myopathy
2	Non-wasting or obese scrapie	Rubbing ; weakness without wasting ; skin lesions	„	6	4	Wasting of muscle, etc., with class 1b lesions but excessive fat deposition throughout body (class 4 lesions)	Severe and widespread myopathy
3	Pining scrapie	Rubbing; marked wasting without ataxia ; skin lesions	„	32	8	Marked wasting of skeletal muscle which has a fish-flesh texture (class 2 lesions)	Myopathy widespread
Group B							
4	Inapparent myopathy (P1) (? preclinical scrapie	No rubbing ; slight unthriftiness only	Unknown	5	5	Occasional "putty" foci (class 1b) without muscle wasting	Localised myopathic lesions
5	Acute hind limb palsy (P2)	No rubbing ; great weakness of hind limbs of rapid onset	Fatal	5	5	Severe degeneration of anterior tibial muscles (class 1a)	Severe myopathy in these muscles
6	*Post-partum* pining (P3)	No rubbing but a nibbling response; severe wasting without ataxia	Many recover	73	2	Severe wasting of skeletal muscle with lesions, mainly of class 2, but occasionally of class 1b	Widespread moderately severe myopathy
7	Acute asthenic paralysis (P4)	No rubbing but a nibbling response ; great weakness of limb muscles	Fatal	3	3	Occasional " putty " lesions (class 1b) with fish-flesh texture (class 2 lesions)	Moderately severe myopathy
8	Juvenile pining (P5)	No rubbing ; general unthriftiness without apparent cause	Unknown	2	1	Wasting of skeletal muscle with lesions of class 2 with some class 1b	Moderately severe myopathy
9	Generalised ataxic pining (P6)	Profound disturbance of muscle movement with wasting but no rubbing or nibbling response	Fatal	1	1	Wasting of skeletal muscle with mainly class 2 lesions	Widespread myopathy
Group C							
10	Amaurotic scrapie	Rubbing with other signs typical of classical scrapie presenting with loss of vision	„	5	4	No basis for visual defect	(Incomplete)
	Controls	(*a*) Normal		15	15	No lesion found	Myopathy not found
		(*b*) Butchers' carcases		—	24		

time, and they form isolated communities free of the majority of the infectious diseases to which sheep are prone. About half the female lambs are retained as breeding replacements in their own flocks and the less desirable animals, usually those born late, ill-grown, or with certain undesirable breed points in, for example, the fleece, are slaughtered before 18 months of age. The average breeding ewe remains in the flock until she has had at least three or four crops of lambs, *i.e.* is five years old or upwards, exceptionally being retained until 10 to 12 years old. The male progeny in these flocks are sold for breeding by 21 months of age, and it is difficult to follow more than a very few, which go as stud rams into other flocks in the Health Scheme. Many of these stud rams are not kept after they are three years old. It is thus more difficult to trace cases of myopathy in males than in females. The sheep are, in the main, kept in folds on arable crops and are fed extremely well, especially during adolescence. The farms are on a wide variety of soils, although the majority of affected flocks are on the lighter soils of sandy nature or overlying chalk with an annual rainfall of less than 25 inches.

When a diagnosis was made the sheep were brought into hospital for periods of a few days up to six months. They were fed liberally on good quality hay, roots, and a balanced ration of concentrates high in good quality protein (which we have used for the past four years for our pregnancy toxaemia experiments), with water *ad lib.*

Autopsy

Some animals died of natural causes, but the majority were sacrificed at various stages of the disease, usually by injection of an overdose of sodium pentobarbitone, occasionally by exsanguination. Autopsy was begun at once and was completed usually

within 12 hours, always within 36 hours. Special care was taken to prevent struggling and violent movements just before or during death. The endocrine glands, the thoracic and abdominal viscera, and central nervous system were dissected out, weighed, and fixed within one to two hours of death. The skeletal muscles were usually left to " set " for three to four hours before examination, to minimise the shortening and distortion which occurs if specimens are excised soon after death.

Histology

Details of the procedures have been published elsewhere (Bosanquet, Daniel & Parry, 1956a).

Diagnostic Criteria

Apart from exhibiting the clinical signs and pathological lesions enumerated below, none of our cases has shown at autopsy any macroscopic evidence of Johne's disease, serious alimentary helminthiasis, fascioliasis of the liver, chronic inflammatory disease of the liver, kidneys, endocrine glands, the nervous system, central or peripheral, or other organs.

Control Animals

Fifteen clinically normal healthy ewes of similar age, parity, and state of pregnancy to the majority of affected sheep were sacrificed and examined carefully at autopsy. They were drawn from flocks with no known history of scrapie for several years. No lesions of the skeletal muscle and adrenals were found, and all the other tissues appeared to be normal with organ weights within the normal range. The skeletal muscles of fore and hind quarters of butchers' carcases have also been examined.

Results

Clinical features of myopathy

Sheep affected with myopathy exhibit a wide variety of clinical signs. There is a progressive loss of power of the skeletal musculature, usually with considerable wasting of the muscles. Disturbances of gait and posture are seen, which are closely correlated to the affected muscles, but without any conspicuous decrease in the tendon reflexes. In addition, many animals rub themselves or scratch with their hind legs, although the usual causes of skin irritation are absent. The disease is almost always slowly progressive and fatal, but we suspect that occasional cases may become stationary or even regress. It is convenient to describe the disease in three stages.

Early signs

The first signs appear at irregular intervals, some weeks apart or under conditions of unusual stress. For example a sheep driven out of the fold as usual will suddenly collapse in its tracks, recovering after a minute or so. It shows only slight ataxia, usually slight clumsiness of the hind limbs, and the head may be slightly raised at rest. In others the ears may flop excessively when moving. There may be a transient disturbance of behaviour, as when a sheep, which has hitherto been docile, refuses to be driven and runs at the dog, or at closed gates. This disorder may last for half an hour or less, and may be absent on succeeding days. There is often, as yet, no evidence of rubbing or ill-health. In other sheep the first sign is swellings of the ears, due probably to haematomata, which heal, leaving a " cauliflower " ear. In yet others it is a whitening of the tip of the fleece, which appears open and harsh; this preceeds actual rubbing. There may also be slight swelling of the muzzle and face, a " fixed " stare with jerky movements of the eyes, and occasionally defective vision and even complete blindness. Later, many of the same animals begin to rub parts of the body against posts or similar hard objects. The principal site is usually the buttocks, the lower thighs, or the base of the tail, and is bilaterally symmetrical. Often the poll and dorsal neck are rubbed, less commonly the neck in front of the shoulder joint and the ribs just below the elbow. Rubbing is at first difficult to detect, since the sheep only do so occasionally for short periods, and this is easily mistaken for the rubbing practised by many unaffected animals when they are being fed well, or when warm sunshine succeeds showery weather. At this time there may also be a stilted, slightly hypertonic gait, as if the animal were walking on hot cinders. This stage lasts from one to 12 months, usually between two and four months.

Gross clinical manifestations

At this stage the gait is ataxic and in many sheep the hind limbs are placed far apart, while the fore limbs tend to be raised when walking. These signs are much more obvious when an animal is made to run, particularly in a straw yard, in which it has to lift its feet. There is usually, though not invariably, evidence of rubbing at the sites mentioned previously; this may have been so severe that the fleece begins to fall out in patches, but the wool grows again and there is no general tenderness of the staple of the fleece over other parts of the body. Some sheep nibble at the haired skin below the knee and hock, scratch as a dog does with ectoparasites, or rub the bridge of the nose. Over these haired portions of the skin a papular rash is common, and may be associated with swelling of the bridge of the nose.

There is now a progressive loss of health, with difficulty in walking and increased fatigue on moderate exercise. The gait becomes very clumsy, especially if the sheep is hurried. The appetite is apparently maintained although individual food consumption records are not available. While such animals go to the food trough eagerly, they tire readily and do not eat as long, or (probably) as much as their normal colleagues. Rarely, there is neither loss of weight nor of condition.

There is no gross abnormality of the nervous system. The postural and tendon reflexes are normal, though the patellar reflex may be somewhat weak in advanced cases. Response to pinprick is within normal limits, and no constant disturbance of cutaneous sensation has been found. The ataxia is not markedly changed by blindfolding. Light pressure between the fingers in the area of the buttocks, base of the tail, dorsal lumbar region and elsewhere elicits a " nibbling " response. In this behaviour pattern the animal

raises its head and nose, and makes nibbling and smacking movements of the lips with extrusion of the tongue, the whole attitude suggesting pleasure. It is difficult to obtain this response by light touch over rubbed areas, but it may be readily elicited by deep muscle pressure, particularly along the back and over the semimembranosus and semitendinosus muscles. If local anaesthetic be infiltrated subcutaneously, the response cannot be elicited over the site for some hours. Defaecation and micturition, righting reflexes, head and neck postural reflexes, eye reflexes, and deglutition are essentially normal, although some cases have difficulty in prehending food and swallowing. A few have an abnormal bleat. In occasional cases there are signs of disturbances of alimentary motility with recurrent ruminal and, more rarely, intestinal tympany. The stools are normal. Rumination may be less frequent but this is difficult to assess with certainty.

Palpation of the skeletal muscles is difficult because of the fleece. There is usually considerable wasting and flabbiness in the dorsal lumbar muscle mass, although in some cases this is obscured by the large amount of subcutaneous and intermuscular fat; in other cases these muscles are firm and even hard.

This stage may last two weeks to two months, usually about four to six weeks.

Terminal stage

Wasting is profound and the sheep looks like a living skeleton; there is frequent rubbing, gross ataxia, ready fatiguability with the slightest exertion, and disturbances of behaviour. The stance is wide-based with over-flexion of the limbs, so that the sheep usually moves at a slow gallop, or the hind legs are moved together. It is usually unable to walk more than 50 to 100 yards at a time, and may not be able to raise its head off the ground for more than two minutes. If such animals are bustled they may go down and never regain their feet, and they are very upset if their fold mates are removed, or if they are changed to new surroundings. The animals continue to feed and suckle their lambs. Eventually they are unable to rise to their feet unaided, but if housed indoors they may live for several weeks lying on their sides but usually with hypertonicity of the limbs, so that they cannot be maintained in a position of sternal decubitus. They die of progressive inanition and exposure, usually during a cold, wet night.

Morbid Anatomy and Histology of Myopathy

As a detailed account is given elsewhere (Bosanquet, Daniel & Parry, 1956a), only the principal findings will be mentioned here. As many other observers have noted, there are no constant lesions to be seen at *post-mortem* examinations as usually carried out. If, however, the skeletal musculature be carefully dissected, lesions of varying extent and severity will invariably be found in many muscles. The degree of wasting can be gauged roughly by weighing the carcase flesh and the bones, and expressing these weights as a ratio of flesh to bone. The adrenal glands have also constantly been enlarged in the late stages of our cases of the disease. The heart may be affected.

In assessing the colour of affected muscles, care has been taken to compare them with the muscles of normal sheep of similar age, dying or killed in a similar manner. The skeletal muscles of the normal adult sheep vary in colour from the deep red of the biceps femoris, medial head of the triceps, or the external vastus, to the lighter red of the coracobrachialis, vastus intermedius, plantaris, etc. Between these extremes are muscles intermediate in colour, such as the semimembranosus, semitendinosus, teres major, and iliopsoas. All the muscles of lambs are, of course, much paler than those of adult sheep.

The myopathic lesion occurs in a number of different forms, which may be described conveniently as follows. These lesions in the main affect the same muscle on each side of the body to about the same extent, but there are instances where the muscle on one side of the body is much more severely affected than that on the other.

1. *Diffuse and focal lesions of intense pallor* (class 1). These lesions which are cream-coloured like putty may affect one or more muscles completely (diffuse lesions, class 1a) (Fig. 1), but more commonly occur as foci, 2 to 5 cm. in diameter, widely disseminated throughout the skeletal muscles (disseminated focal lesions, class 1b) (Fig. 2). The foci are sharply demarcated as a rule. In some cases the affected focus is raised slightly above the adjacent unaffected muscle as if there were a pseudo-hypertrophic phase (class 1c lesion) as in some human cases of muscular dystrophy, and occasionally have a flecked appearance like a thrush's breast (class 1d lesion).

Diffuse lesions have been seen in widely separated muscles, *e.g.* the semimembranosus, the semitendinosus, the dorsal straight muscles of the neck, the anterior tibial, tensor fascia lata, external obturator, etc. Disseminated focal lesions may be found in most muscles. Thus foci may be confined to the lower parts of digital flexors and extensors, the muscles adjacent to the shoulder and hip joints, the scapular muscles, the ventral throat muscles, and the deep spinous interosseous muscles of the vertebral column; the diaphragm and heart are occasionally affected. The only way in which one can be certain that the odd focal lesion is not missed is to dissect out and make multiple incisions in each individual muscle in the body. This technique has been carried out on the animals discussed here.

2. *Generalised wasting or pining* (class 2 lesions). The mass of the skeletal muscles is greatly reduced, the muscles are paler than normal and translucent, rather like those of fish. Such muscles are more flabby than usual, rarely show pronounced *rigor mortis*, and do not "set" well. Individual muscles may be reduced to half their usual weight. Foci of moderate pallor, light pink in colour, occur in numerous muscles, but the sharply demarcated "putty" lesions are rarely seen. The atrophy is sometimes much greater in individual muscles such as the iliopsoas, the facial and auricular muscles,

external eye muscles, and muscles of the larynx, pharynx, and tongue.

3. *Firm lesions* (class 3). This type is less common and has been confined to the sternomastoid, scalenus, and ventral vertebral muscles adjacent to the thoracic inlet. The muscles are firmer than normal, rose-pink in colour, and are usually affected throughout, although the sternomastoid may be affected only where it is adjacent to its thoracic attachment.

4. *Excessive deposition of fat* (class 4 lesions). The muscle appears to be normal from the surface, but when incised, it is found to be largely replaced by fat. Those most commonly affected are the biceps humeris and abdominal muscles; the latter may give way, producing what is termed a "broken-bodied" ewe with pendulous lower abdomen. In this type of myopathy there is usually excessive inter- and intra-muscular fat, so that muscles appear to be "marbled" like prime beef, but the fat is more liquid at ordinary temperatures than normal mutton fat.

Histology

The signs of a primary degeneration of the muscle fibres are profound and widespread in portions of muscle showing macroscopic lesions (see Figs. 3, 5, and 7), while adjacent portions of normal muscle may show no abnormality. In mild cases one or more muscle fibres only may be affected in a field, they are wider than normal in longitudinal section, and in transverse section they are rounded instead of polygonal in outline, and their transverse striations are indistinct (Fig. 5). In other lesions many fibres may be affected, until in severe lesions only an occasional fibre is left intact (Fig. 7). In these more advanced lesions the sarcolemmal nuclei are increased in number and many nuclei appear in the centre of the fibre; short lengths of the muscle fibre are fragmented and the sarcoplasm may be granular (see Fig. 3). There is no perivascular cuffing, and interstitial infiltration of cells is infrequent; no collections of polymorphonuclear cells such as might suggest an inflammatory lesion have been found. Calcification has never been seen.

The distribution of the degenerated fibres within a bundle has been irregular and is quite distinct from that seen in muscle degeneration secondary to interruption of the nerve supply to the muscle.

These lesions are very similar to those seen in human dermatomyositis (polymyositis) and muscular dystrophy (see Bosanquet, Daniel & Parry, 1956a).

Sarcocysts have been present in almost every animal we have examined, myopathic and normal.

A preliminary examination of the central and peripheral nervous system has revealed no conspicuous constant lesions, apart from occasional vacuoles in the cytoplasm of some of the larger neurones of the brain and spinal cord.

Classification of the Principal Syndromes

The clinical forms of the disease show a large range and variety, but they can be classified for descriptive purposes into syndromes, in which we have observed remarkably good agreement between the clinical signs and the principal muscle lesions. These are summarised in Table I. The syndromes fall into two groups: A, in which rubbing is severe, and B, in which no rubbing is seen.

Group A. Syndromes with rubbing

The prominent signs in this group are rubbing and scratching of the wool portions of the body and the poll, and nibbling of the haired portions of the lower limbs with a well-marked nibbling response. Progressive weakness (asthenia) and severe emaciation usually follow; the disease is probably almost always fatal. These syndromes are normally considered to be scrapie.

1. *The asthenic ataxic type* (classical scrapie). There is gross ataxia with a wide-based stance particularly of the hind legs, early fatigue on exercise, and finally great weakness with only moderate loss of flesh. At autopsy, "putty" lesions, both total (class 1a) and disseminated foci (class 1b), are prominent. The flesh-bone ratio is usually 1.5-2:1. In two animals unable to raise their heads off the ground for longer than a minute, total "putty" lesions in the dorsal straight muscles of the neck were found.

2. *The asthenic non-cachectic obese type* (non-wasting scrapie). There is progressive weakness with excessive fat deposition, so that affected animals maintain or even increase their bodyweight. Eventually they become too weak to rise. At autopsy there is a great reduction of the skeletal muscles and a corresponding increase of subcutaneous and intermuscular fat, together with disseminated "putty" foci (class 1b lesions) and intramuscular fat (class 4 lesions). The flesh-bone ratio is 2-3:1.

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FIG. 1.—Lateral view of upper left hind limb of a sheep affected with scrapie. Note the extreme pallor of the semimembranosus and semitendinosus muscles (class 1a lesion); the incision in the latter shows that the whole depth of the muscle is pale. The biceps femoris and adductor muscles are the normal dark (red) colour. The white tissue is fat and fascia. × 0·25.

FIG. 2.—View of part of a fore limb from a sheep with scrapie, showing the coracobrachialis muscle incised to show "putty-coloured" myopathic foci throughout the muscle (class 1b lesions). Note normal dark muscle above and to left below. × 0·4.

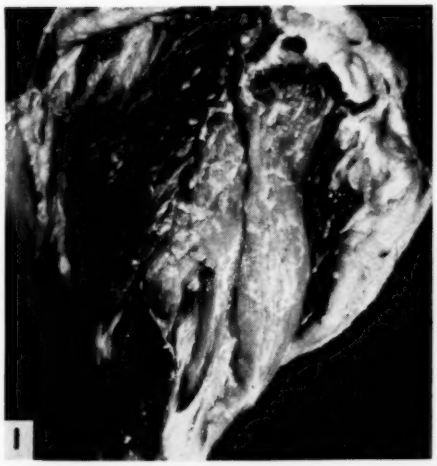
FIG. 3.—Longitudinal section of a portion of a myopathic anterior tibialis muscle from a sheep with hind limb palsy. Note the normal striated muscle fibre above, the swollen disintegrating fibres below and the numerous nuclei. × 370.

FIG. 4.—Longitudinal section of skeletal muscle of a normal sheep. Note the regularity of the fibres and their striations and modest density of nuclei. × 280.

FIG. 5.—Longitudinal section of skeletal muscle with myopathy at an early stage from a sheep with scrapie, for comparison with Fig. 4. Note the loss of cross striations and the corrugation of the muscle fibres, with an increased density of nuclei. × 280.

FIG. 6.—Transverse section of skeletal muscle of a normal sheep showing a muscle bundle. Note the polygonal outline of the individual fibres and the small number of nuclei. × 216.

FIG. 7.—Transverse section of a skeletal muscle with myopathy at a late stage from a sheep with scrapie, for comparison with Fig. 6. Note replacement of muscle fibres by a mass of small nucleated cells. The few recognisable muscle fibres are enlarged with rounded outline and granular sarcoplasm. × 216.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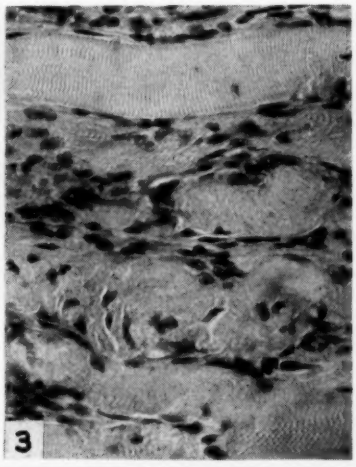
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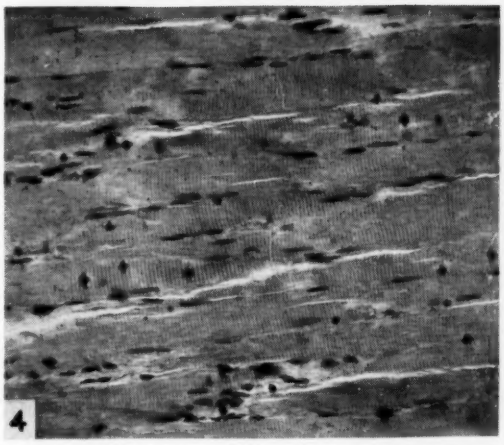
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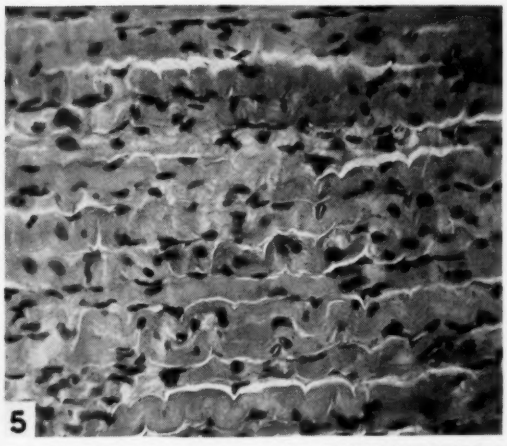
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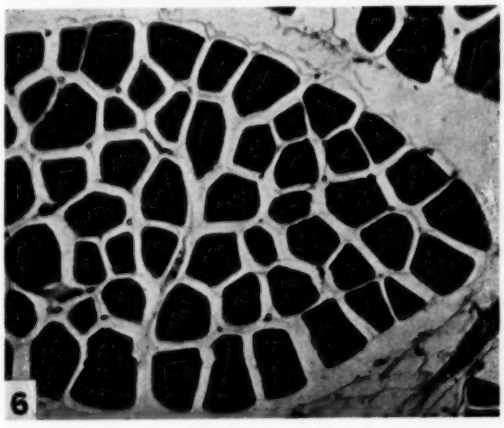
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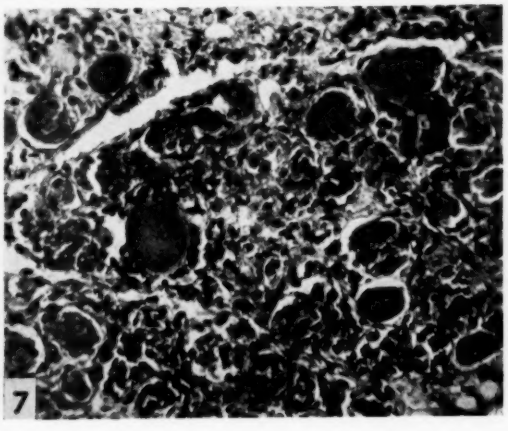
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3. *The asthenic cachectic type* (pining scrapie). There is progressive and severe emaciation without ataxia until near death. At autopsy there is a generalised atrophy of the skeletal muscles (class 2 lesions) without " putty " lesions but often accompanied by firm lesions (class 3 lesions) of the ventral neck muscles. The flesh-bone ratio is 1.0-1.5:1.

Group B. Syndromes without rubbing

These syndromes have occurred, sometimes in considerable numbers, in flocks from which cases of group A syndromes were drawn.

4. *Inapparent Myopathy* (P1). These sheep are apparently normal, although they may exhibit some vague unthriftiness, which leads a shepherd to cull them. Many are the offspring of scrapie sheep, but have not reached the age when clinical signs of the disease usually appear. Foci of " putty " degeneration (class 1b lesions) are found in a few situations, while the bulk of the skeletal musculature is normal.

5. *Acute hind limb myopathic palsy* (P2). Affected animals become unsteady on their hind limbs, their hind fetlocks give way and flex under them as they walk, and in 7 to 10 days they are unable to rise on their hind limbs. They are able to stand up on their fore limbs and their head posture is normal. In the terminal stages their hind limbs are kept extended behind them. At autopsy severe lesions (class 1a) were found in the anterior tibial group of the digital flexor muscles of the hind limbs in all of five cases. Four were affected one to three weeks after lambing, the fifth before lambing; parturition and the early puerperium were normal.

6. *Post-partum myopathic pining* (P3). This condition usually develops during lactation, more rarely in late pregnancy. There is profound wasting of skeletal muscle and weakness, but no loss of appetite or ataxia. Many animals recover. At autopsy on two cases typical generalised pallor (class 2 lesions) of the skeletal muscles with occasional " putty " foci (class 1b lesion) were found. These sheep are commonly termed " piners " or " wasters " by shepherds.

7. *Acute myopathic asthenic paralysis* (P4). Sheep affected with this type of the disease are usually in excellent bodily condition and apparently in excellent health, when they develop suddenly, in a few hours, great weakness, so that they are unable to rise to their feet. This may be preceded by some episode with excitement. If lifted up they can walk, but their ability to maintain a standing posture is much impaired, usually due to weakness of one or other hind limb. When lying they usually do so on the side of the weaker limb and there is hypertonia of the limbs so that it is very difficult to maintain them in sternal decubitus. With suitable nursing they feed well and show none of the mental changes usually present with types 1 to 3. These sheep generally have a skin rash on the ears and sometimes the face, but they do not rub, although a nibbling response can usually be elicited by deep digital pressure across the loins. At autopsy typical disseminated foci of degeneration (class 1b lesion) are present, notably in the deep muscles around the hip joint, with more generally distributed class 2 lesions.

8. *Juvenile myopathic pining* (P5). Only two cases of this type have been recognised to date, one of which has come to autopsy and in which all the macroscopic (principally class 2 lesions) and microscopic lesions of myopathy have been found. It is first noticed in adolescent animals three to nine months old, which for no apparent reason become very unthrifty with a dull dead fleece, and fail to grow normally. These cases do not rub and no nibbling response has been present. In the later stages the animals are weak and emaciated, and lie about a great deal, although they feed fairly well.

As this type has only just been recognised, it seems probable that it may be more common than it might appear to be, since cases of this kind would usually be attributed to helminthiasis or faulty nutrition. The recognition of this type is of considerable interest in any consideration of the natural history of these myopathies.

9. *Generalised myopathic ataxic pining* (P6). This type is very similar to type 1 (classical scrapie) except that signs of rubbing and the nibbling response are absent.

The clinical signs are gross ataxia of the limbs, disturbances of eye movements and head posture, general weakness, and wasting of the skeletal muscles.

Group C. Syndromes with specific neurological defects

10. *Amaurotic scrapie.* In a series of five cases, all of which developed scrapie of types 1 or 2, the presenting sign was the sudden occurrence of defective vision which quickly led to apparently complete loss of vision. The pupillary light reactions and the eye preservation reflex were then absent. The media, fundus, and disc were normal. No macroscopic abnormality of the visual system was found at autopsy; a detailed microscopic study has not yet been made.

Relation between clinical signs and myopathic lesions

In most cases of the disease the clinical weakness and ataxia may be explained by the widespread distribution of the myopathic lesions and the severe involvement of some muscles with sparing of their antagonists.

In certain cases where the myopathy is particularly severe in certain muscles with special functions, it is possible to relate the clinical signs to the myopathy very clearly. Thus the cases of hind limb palsy have all had complete pallor of the anterior tibialis muscles; cases with drooping ears showed atrophy of the auricular muscles; cases with abnormal eye movements showed myopathy of the external ocular muscles; cases with difficulty in feeding and swallowing showed involvement of the pharyngeal and tongue muscles; sheep with abnormal voice (bleat) have the laryngeal muscles severely affected; two cases with inability to raise the head for more than one to two minutes showed complete pallor of the dorsal neck muscles between axis and occiput, and two cases with pendulous abdomen, so-called " broken-bodied " ewes, had severe lesions in the dorsal abdominal muscles and tensor fascia lata of the affected side.

Myopathy and Scrapie

In all of 48 consecutive cases of scrapie, in which careful autopsy examination of the muscles has been carried out, we have found definite macroscopic lesions of a *widespread* myopathy, which have been confirmed by histological examination. As myopathic lesions have been present in all our natural cases of scrapie, examined properly, it seems likely that scrapie is but part of a more widely occurring group of myopathic diseases.

Epidemiology

This group of myopathic disorders is widespread. The highest mortality rate we have observed was 26 per cent. in one flock of 105 breeding ewes in one year, but rates of 2 to 5 per cent. in moderately affected flocks for a series of years are not uncommon, while in other flocks only very occasional or suspicious cases occur. Both males and females are affected, but our data are insufficient to establish whether there is a sex predisposition, as so many more females than males are under observation. In both males and females most cases are two-and-a-half to three-and-a-half years old (early middle age), although fatal cases develop as late as seven and eight years old. Second-crop females are particularly prone to the disease and 25 to 40 per cent. of this age group may be lost annually in severely affected flocks. New cases occur at any time of the year but are much more prevalent from mid-pregnancy onwards, and particularly after parturition, *i.e.* during the early winter and early spring.

The disease tends to occur in the progeny born in certain flocks, and hence with a common parentage and environment. It may suddenly appear in a severe form when the progeny of a certain mating have their second pregnancy. Thus in a 200-ewe flock hitherto free of cases of scrapie for at least four years, 23 per cent. of the second-crop ewes suddenly developed the disease in one year; 86 per cent. of these ewes were sired by one ram out of ewes by another ram. Not all the progeny of affected dams are affected, but the granddaughter of an affected ewe may be affected while her dam was unaffected.

The disease appears to be more prevalent in certain districts with characteristic soils, but adequate proof is hard to obtain. Of 107 shearling (one-and-half years old) ewes sold from one flock to two receiving flocks, 15 per cent. and 10 per cent. respectively of these ewes in the receiving flocks developed the disease with their second pregnancy while the ewes of similar age and breeding retained in the original flock remained healthy. Sheep from well-managed flocks going to less well-managed flocks, particularly if the general management and plane of nutrition are lower, seem more liable to develop the disease. The disease is apparently not spread from affected to unaffected ewes by the ram at coitus, nor by contact with affected animals. The natural disease occurs in many breeds of British sheep and their crosses; I have first-hand information on six pure breeds and four crosses, which include short- and long-wooled breeds, black- and white-faced sheep, lowland and mountain sheep.

The course of the disease in individual animals varies from one to six months, but is usually two to three months.

Economic Importance

It is impossible to do more than guess the annual cost to British agriculture, for no figures for the losses from this group of myopathic diseases are available. In many folded flocks the losses have been such that flockmasters have given up keeping self-contained breeding flocks on account of the disease. The decline in sheep numbers in the arable counties since the 1930's is only in part due to the impact of rising costs, the 1939-45 war, and the scarcity of shepherds; in many instances the decisive factor has been the recurrent annual loss from these myopathic diseases, more particularly from scrapie.

In lowland breeding flocks it is usually considered that the total loss of breeding ewes should not exceed 5 per cent. per annum, if a self-contained flock is to remain profitable. In well-shepherded flocks this annual mortality rate may be as low as 2 to 3 per cent. from all causes other than the myopathic diseases. When, in addition to this "normal" mortality rate, losses from myopathy add a further 3 to 5 per cent. per annum, the flock no longer remains an economic proposition, owing to the short productive life of the females and the excessive cost of replacements.

It seems not improbable that the annual loss is at least £100,000. Many flocks lose 5 to 10 per cent. of their breeding females annually, and with 5,000,000 lowland breeding ewes an estimated incidence of one case in every 500 ewes is probably low. This figure gives 10,000 new cases each year, on which the average loss is about £10 per head. Quite as important is the psychological effect on flockmaster and shepherd of continuing insidious losses over which they have no control, and which give rise to apathy and a general lack of interest in the flock.

Differential Diagnosis

It is often extremely difficult to make a clinical diagnosis in the early stages of some forms of myopathy and a thorough knowledge of the health history of the flock for some years is of the utmost value. It is only practicable to mention the principal syndromes with which the myopathic diseases may be confused:—

1. The rubbing cachectic types presenting at or shortly after parturition may be very similar to mild cases of toxaemia of late pregnancy, in which a tenderness of the fleece occurs with rubbing and varying degrees of wasting. These latter cases usually do not show a nibbling response, but it may be wellnigh impossible to distinguish these post-toxaemic cases from scrapie unless they are under observation for six to eight weeks.

2. The post-partum pining (P_3) and juvenile pining (P_5) show unthriftiness and wasting similar to that seen in alimentary helminthiasis, cobalt deficiency, general malnutrition, and chronic infections, and these disorders must be considered first and be excluded.

3. The acute posterior palsy (P2) and the acute asthenic paralysis (P4) may simulate traumatic injury, either at parturition or from external agencies, etc., but evidence of trauma, damage to the spinal cord or peripheral nerves is lacking and the limb reflexes are virtually normal.

4. Sheep are affected by two main types of dietetic muscular dystrophy. In the first, called "White-Muscle" or "Stiff-Lamb" disease, the literature on which has been reviewed recently by Blaxter & McGill (1955), there are grey-white firm lesions in the skeletal and myocardial muscles. The disease affects sucking lambs principally, notably those without access to fresh green foodstuffs (Vawter & Richards, 1939; Cotchin, 1948). The disease can be prevented and mild cases cured by vitamin E therapy (Sholl, 1939; Willman *et al.*, 1946). The clinical history, epidemiology, macroscopic appearances, and therapeutic response of this vitamin E controlled muscular dystrophy are quite unlike those of myopathy.

The second type is more akin to the paralytic myoglobinurias of horses and cattle. It appears suddenly after severe muscular exertion and is accompanied by redwater. Death may occur in a few hours, while milder cases often gradually recover. A syndrome of this type has been described recently by Marr, Sharman & Blaxter (1956) in hoggs in the north of Scotland. This type appears to be quite distinct from our myopathy with its slow, insidious, progressive course, no redwater, and no history of unusual exertion. The possible relation of these two types of muscular dystrophy to our myopathy have been discussed more fully elsewhere (Bosanquet, Daniel & Parry, 1956b). They are quite distinct from our myopathy.

5. "Weisses Fleisch" is a term applied in Germany to pale lesions seen occasionally in skeletal muscle of sheep carcases. Examples of what are presumably this lesion have been seen in the butchers' carcases of otherwise normal sheep; the lesion is larger, contains more fat, and is dense rather than translucent in texture, which distinguishes it clearly from a myopathic lesion.

6. Infected and/or infarcted foci of muscle show patchy congestion and areas of brown and yellow pigmentation with exudation and/or a firm consistency of the muscle.

Treatment

No treatment is known which will arrest and reverse the disease, except P3, once frank clinical signs have appeared. It is possible that the progress of the disease may be retarded by careful nursing and good feeding if animals are housed in individual pens in a warm, draught-free building.

We have tried, in addition to good nursing and feeding, each of the following treatments on one or two animals, but without any sign of modifying the expected course of the disease:—

Vitamin B_1 given subcutaneously as 1 g. per day of aneurine hydrochloride in aqueous solution for two months.

Vitamins B_1, B_2, B_6, and nicotinamide given as a combined preparation* intramuscularly which provided 20 mg., 8 mg., 8 mg., and 80 mg. respectively of the four vitamins twice daily for six days.

A supplement of high-quality protein, minerals, and accessory food factors fed in the form of dried liver† and spray-dried whole milk powder at levels of 37 to 75 g. and 200 to 300 g. respectively for 40 to 50 days.

Vitamin E as α-tocopheryl acetate given subcutaneously as 350 mg. in arachis oil daily for 32 days and thereafter 200 mg. daily *per os* for 20 days.

Vitamin B_{12} given subcutaneously in doses of 2 mg. daily for four days and thereafter 250 μg. daily for 15 days.

Desoxycorticosterone acetate given subcutaneously as 15 mg. in oily solution daily for 35 days.

Progesterone given intramuscularly as 20 mg. in oily solution daily for 50 days.

Adrenocorticotropic hormone given subcutaneously in a dose of 40 I.U. twice weekly for three weeks.

Three five-day courses of British anti-lewisite (B.A.L.) with an interval of two weeks between courses.

Neostigmine in single doses of 2.5 to 5.0 mg. intramuscularly may sometimes provide a slight, very transient improvement in the early stages.

The results of these pilot therapeutic trials suggest that the disease is not a peripheral neuropathy due either to a deficiency of the vitamins of the B-complex or to a chronic intoxication with heavy metals such as arsenic, which might be expected to be relieved by B.A.L.; that it is not a muscular dystrophy of vitamin E controlled type nor a general dietary deficiency disease; that it is not an endocrine disturbance readily amenable to the replacement therapy given, nor a primary neostigmine-alleviated disorder of the myasthenia gravis type.

Discussion

The discovery of a spontaneous degeneration of skeletal muscle (myopathy) in the sheep opens up a new field in studies of animal disease, for a widely occurring spontaneous myopathy of this kind has not, as far as we can ascertain, been described before in domestic animals. This myopathy is quite distinct in its macroscopic pathology, epidemiology, and therapeutics from "stiff-lamb" or "white-muscle" disease of sheep (Hobmaier, 1926; Sholl, 1939; Willman *et al.*, 1946) which has been reported in England (Cotchin, 1948), and from the muscular dystrophy of adolescent sheep reported by Marr, Sharman & Blaxter (1956) in Scotland; and it would appear to be different from the majority of cases of "weisses fleisch" (Ziegler, 1929; Seifried, 1944), which we have encountered sporadically in butchers' carcases.

The finding of the myopathy in every one of 48 consecutive clinical cases of scrapie suggests that many, if not the majority, of the natural cases of the disease are in fact cases of myopathy. Indeed, the myopathy provides for the first time a satisfactory

* Becosym.

† Hepavite.

explanation of many of the prominent clinical signs of scrapie. Thus the ataxia, the asthenia, and the wasting, seen in so many cases, are readily explained by the changes in the power and mass of the myopathic skeletal muscles, especially when we realise that the wasting cannot be ascribed to an inadequate food intake. The variation in the degree and type of the ataxia may be attributed in part to the disturbance of co-ordination due to the varying involvement of different muscles in different cases and in part to the degeneration of intrafusal muscle fibres and the muscle spindles, which are now known to play such an important part in the normal maintenance of muscle tone (see Bosanquet, Daniel & Parry, 1956). Indeed in the clinical forms of the disease with special localising signs it is possible to correlate the clinical signs and the principal pathological lesion very closely.

In flocks where scrapie occurs, other kinds of chronic ill-health and disability may occur and in some years these may be more important causes of loss than scrapie. These syndromes have not been recognised previously as far as I am aware, and indeed only came to light when flockmasters and shepherds were encouraged to keep complete records and discuss every case of ill-health and disability occurring in their flocks. It then became apparent that a number of syndromes were occurring to which no definitive diagnosis could be given, and it was only possible to define these precisely when they were found to have myopathy. In this way the syndromes of acute hind-limb myopathic palsy, *post-partum* myopathic pining, juvenile myopathic pining, generalised myopathic ataxia, and acute asthenic myopathic paralysis were recognised. The occurrence of these hitherto unrecognised syndromes in close association with scrapie explains the bewildering confusion among many experienced sheepmen as to what is and what is not scrapie, and the assertion one hears not infrequently that scrapie has several forms which have changed and are changing.

On the information at present available it seems imperative that we change our concept of the nature of scrapie. No longer can we consider scrapie to be a single disease entity; it is but part of a more widely occurring myopathy, several types of which conform to the disease usually called scrapie. We are thus faced with the wider problem of myopathy, rather than the more limited problem of scrapie. It may be that some natural cases of scrapie are not myopathic and that the experimentally induced scrapie differs from the natural disease described in this paper. Indeed, it is possible that scrapie represents a syndrome elicited by several causes in a similar manner to canine-hysteria and epilepsy in the dog which represents the final expression of disordered brain function which may be initiated by a wide variety of agents (see Parry, 1949). But until much more evidence is forthcoming for such contentions, we must assume that all forms of natural and experimental scrapie have a common aetiology.

The aetiology of this myopathy remains uncertain, but some guidance may be obtained from comparing it with analogous human diseases. The sheep myopathy has certain points of similarity with the human diseases of muscular dystrophy and dermatomyositis (polymyositis) (see Bosanquet, Daniel & Parry, 1956; Anon, 1956). These are obscure diseases, which are extremely difficult to treat and are rarely cured. Of the aetiology of dermatomyositis nothing is known, but muscular dystrophy has a definite familial tendency and many forms of the disease are recognised as being hereditary and are inherited as sex-linked dominant, autosomal recessive and intermediate types.

At the same time the evidence we have from the sheep is difficult to reconcile with the theory of an infectious communicable agent with a very long incubation period affecting the central nervous system like the virus of poliomyelitis, and not influenced markedly by the genetic constitution of individual animals. The usual stigmata of fatal virus infections of the nervous system are absent in microscopical preparations, the vacuoles seen in the larger neurones of the brain stem and spinal cord and thought to indicate such an infection are inconstant and occur in animals not affected with scrapie, and in man occur in a number of chronic diseases, while the data suggesting the experimental transmission of the disease are open to question on the grounds that the sheep used in the experiments are not known to be free of the myopathic trait, and in many instances were of a breed well known to suffer severely from scrapie. Many aspects of the epidemiology of the disease are very difficult to explain on the communicable virus theory of causation. The balance of the evidence at present favours an hereditary basis of the disease rather than an infectious one.

The hypothesis, which seems to provide the most likely explanation of all the available facts, is that myopathy (including scrapie) is primarily an hereditary disease controlled by a number of autosomal genes with or without modifying genes, and that the expression of the genotype may be influenced markedly by environmental factors, such as nutritional levels at various stages of growth, the precise nature of the foodstuffs, general management, movement to a new environment, pregnancy, and sexual activity, etc., which modify the general metabolism of the animal. The rubbing and nibbling response may well prove to be a secondary phenomenon due to the widespread disturbance of the normal metabolism of affected animals and possibly related to disordered sensation in the degenerating intrafusal muscle fibres and their proprioceptive receptor organs.

These observations raise many new questions, which it is quite impossible to answer at the present time. Thus one would like to know more about the cause of the nibbling response, the rubbing, and the changes in mental behaviour, for which no one has yet advanced satisfactory explanations; why some scrapie sheep waste and why some put on excessive fat; the relationship between the different forms of myopathy and what factors determine which muscles are affected and how early in life this occurs; the factors governing the development of the disease and the possible rôle of environmental and special precipitating causes, for we know there are inapparent

forms, some of which may probably never show obvious clinical signs; the nature of the early stages of the muscle defect; the cause of the blindness seen in some cases, for which no morphological basis has yet been demonstrated; the nature and cause of the skin rash and of the frequent swellings of the ears; and in what way the vacuolation of neurones may be related to the myopathy (three of the four non-scrapie control sheep showing vacuoles in Holman & Pattison's (1943) series were suffering from pining); and the precise nature of the experimental disease produced by intracranial inoculation and its relationship to the naturally occurring disorder.

The Investigation and Attempted Control of Scrapie

That our knowledge of the natural history of scrapie and its related myopathic diseases has been so fragmentary, is largely due to the failure of the veterinary scientist to obtain and retain the confidence of a large enough group of flockmasters and shepherds. Sheepmen are extremely reluctant to admit to any knowledge of the disease, let alone allow that they may have occasional cases in their own flocks. The mere visit of a veterinary surgeon in his capacity as an investigator of disease is usually sufficient to dry up the sources of information.

With the occurrence in 1952 of scrapie in British sheep exported from the United Kingdom to Australia two years previously, and the subsequent imposition of a complete embargo on the importation of all sheep from the U.K. into Australia, it seemed essential that the problem be tackled anew. The first step was to ascertain what the disease was, where it was, and how important it might be. After consultations with a number of experienced sheepmen, it became clear that the only practical approach was to enlist the support of a group of the leading flockmasters and shepherds under conditions which allowed one to guarantee that any information they provided would be treated in the strictest confidence and that suitable steps would be taken to guard against any leakages of this information. This has been done and the collaborating flocks organised with the help of the Suffolk Sheep Society as members of a Voluntary Sheep Health Scheme, the aim of which is to increase the productivity of member flocks by ascertaining and, wherever possible, reducing causes of loss, while at the same time building up a system of flock records, checked twice a year by an independent person, which may serve in the future as an indication to prospective purchasers of the general health of the flock. Members pay an annual fee and in return receive general supervision and advice on their flock management; they undertake to organise their flocks so that individual records are kept of all animals, of their health and production, and of all causes of ill-health, disability, and death. Members are encouraged to consult their veterinary surgeons as much as possible and to enlist their aid, as no curative or prophylactic treatments are provided by the Health Scheme, which, of course, is also not concerned with the statutory diseases normally dealt with by the Animal Health Division of the Ministry of Agriculture, Fisheries and Food.

The results of four years' experience of the Scheme have proved most encouraging and have provided a working knowledge of the pitfalls as well as advantages of such an approach to the problems of animal health and production. In the early years, losses from toxaemia of pregnancy were the principal problem, but these have been virtually eliminated; there were only five fatal cases among some 5,000 ewes in the 1956 lambing season. Infestations of parasite worms are rarely of any importance. Other diseases such as entero-toxaemia and contagious pustular dermatitis are no longer a problem. The control of these other causes of loss has thrown into relief the prevalence of scrapie and related myopathies and is providing much valuable information about the precise occurrence of the various syndromes in different families within a breed. At the same time a number of other possible hereditary traits are under scrutiny, and the results are being used wherever possible as a basis for long-term breed improvement. The data which are being provided by the Scheme should be of increasing value for research as pedigree and production records for longer periods become available.

The information already obtained from these records and summarised in a preliminary form in this paper changes the whole emphasis of my approach to scrapie. No longer can scrapie be looked upon as a single disease; rather must we consider it as one of a group of myopathic disorders which have at least a common pathology. The important question thus becomes not whether a flock or breed is free from scrapie, but whether it is free from myopathy. There are, as far as I am aware, no flocks which could provide suitable records over a sufficiently long period to sustain a claim that they were absolutely free of scrapie, let alone myopathy. Thus assertions that flocks or breeds are free of scrapie can hardly be taken seriously. Indeed, scrapie and scrapie-like diseases have been known in many countries of Western Europe for 200 years in both merino and non-merino sheep stocks; it is thus improbable that the scrapie and myopathic trait has not been taken to those countries, whose present sheep stocks have been derived from those of Western Europe during the past two centuries. Under the conditions of extensive sheep-raising such as largely obtained in the Southern hemisphere and much of the North American continent, a low incidence of scrapie is likely to go unnoticed; indeed in the absence of signs of severe rubbing, losses from myopathy would occasion little comment.

Since 1952 scrapie has been recognised officially for the first time in Australia, New Zealand, Canada, and the United States. The first cases recognised were in sheep imported from the United Kingdom or in the offspring of recently imported sheep. However, as the disease receives more attention, it seems that not all cases can be traced to recently imported sheep and their progeny and the data suggest more and more that a low incidence of the disease has been present in some of these countries for a long time but has received little attention. It is perhaps becoming increasingly difficult to sustain the plea put forward

at the time the first cases were recognised, that these large sheep-raising countries are free of scrapie apart from the few cases in recently imported sheep.

There is at present no test which may be used to establish the diagnosis in doubtful cases of scrapie and other myopathies, or to determine potential cases of these diseases in their clinically inapparent stages. We are thus forced to rely on the clinical signs, coupled now with the finding of myopathic lesions at autopsy. A diagnostic test would be of immense value in studying and controlling the disease, but until one is available we have to rely on clinical records. This means having the complete confidence and collaboration of flockmasters and shepherds.

Since the occurrence of scrapie in exported British sheep, there have been various proposals for the control of the disease in the United Kingdom, based in the main on the concept of an infectious virus disease of the usual communicable type with a very long incubation period. An early proposal that scrapie be made a notifiable disease and affected animals slaughtered with compensation was wisely refused by the Ministry on the grounds that it was so difficult to diagnose scrapie in the early stages that any regulations would be most difficult to enforce. A more recent proposal is that farmers with affected sheep should be asked to notify a central authority who would then exercise supervision and impose a quarantine on the sale of all stock until the flock was declared free of the disease. Variations of these two proposals have been adopted in Australia, New Zealand, Canada, and the United States where all the animals in a flock found to be affected are slaughtered, and flocks which have received animals from such affected flocks during the previous two or more years are placed in quarantine and only allowed to sell sheep for slaughter. The ramifications of such a policy can cause very serious dislocation of established farming practice; thus, for example, following two cases of scrapie in imported British sheep, 150,000 sheep are now said to be in quarantine and may only be sold for immediate slaughter, although many would normally be sold more advantageously as breeding stock.

In countries where scrapie has been endemic for many years, a more conservative line of action seems appropriate, at least until such time as data are available to establish the cause of the disease, and, if it should prove to be infective, how infectious the causal agent may be. Control measures should not be more onerous than the disease itself; they should disturb the general organisation of the sheep industry as little as possible, and they must also command the general support of flockmasters and shepherds, for without their whole-hearted support any system of control and eradication is doomed to failure.

In Britain, where the disease has been present for at least 150 years with a general attack-rate of probably not more than 1 to 2 per cent. (although higher attack-rates have obviously occurred in certain localities from time to time), attempts to control the disease have hitherto been entirely in the hands of flockmasters and shepherds who have in general attempted to eliminate the strains which developed the disease. These efforts have been applied empirically, by individual breeders—principally the pedigree breeders. It is virtually impossible (unless he takes very special precautions) for the commercial flockmaster to take any action in preventing the disease, for it is difficult for him to identify his sheep individually, and where females are purchased it is usually impossible to trace their origin, let alone their parentage, two to four years after the date of purchase, which is when the disease is likely to appear.

Bearing these facts in mind, the right approach in Britain seemed to be to enlist the best pedigree flocks in a voluntary scheme which would allow the co-ordination of efforts to control the disease on the existing policy of eliminating scrapie strains, but on a breed instead of an individual flock basis, while reducing other causes of loss of profitability. At the same time the scheme should provide as much epidemiological and scientific data as possible, and yet be in large measure financially self-supporting and independent, which would allow ready modifications in the light of experience. The Sheep Health Scheme referred to in this paper was designed to meet these needs and has received encouraging support. The crux of the Scheme is the mutual confidence between flockmaster and shepherd on the one hand and the scientific adviser on the other, so that each party feels personally responsible to the other for the success of the individual flock and the Scheme as a whole. To obtain such a basis a scientific adviser must be in a position to give advice which is sound economically and practicable agriculturally, and to guarantee that the detailed information given him will not be disclosed to any other individual or organisation without the consent of the giver. It is difficult for Government agencies as at present organised to meet this latter essential requirement.

Should it appear desirable to expand the work on the field studies of scrapie and myopathy and to investigate more intensively measures of control and eradication, the health scheme method seems an admirable instrument. The experience gained in initiating and running the present Scheme would provide a foundation, from which an expansion could be organised to provide separate schemes for each of the principal breeds of sheep. These might be based on regions and attached to the University serving the region, for such a scheme provides a simple, cheap, and effective method of applying preventive veterinary medicine and advanced animal-breeding techniques to the long-term problems of animal production, while at the same time providing a fruitful source of reliable data for scientific investigation.

Summary

1. The existing information regarding scrapie is reviewed. The idea that it may be due to a simple communicable virus infection of the central nervous system is difficult to sustain.

2. New data on the clinical and pathological features of the disease are presented.

3. A hitherto unrecorded degenerative lesion in the muscles (myopathy) of scrapie sheep is described.

This myopathic lesion may take several forms, namely diffuse and focal lesions of intense pallor, a translucency like the flesh of fish, firm pink lesions and excessive fat deposition. The lesion is usually bilaterally symmetrical. The myocardium may be affected.

4. Microscopically, the lesion consists of a degeneration of the muscle fibres, which are enlarged and lose their cross striations, with an increase of sarcolemmal nuclei. Eventually the muscle fibre disintegrates. The muscle spindles may be affected.

5. The location and extent of the muscle lesion is closely correlated with the clinical signs.

6. The myopathy is not confined to scrapie but is responsible for several other syndromes, which occur in flocks affected with scrapie. These syndromes have not been recorded previously and are provisionally termed:—

P1 or inapparent myopathy, P2 or acute hind limb myopathic palsy, P3 or *post-partum* myopathic pining, P4 or acute myopathic asthenic paralysis, P5 or juvenile myopathic pining, and P6 or generalised myopathic ataxic pining. These syndromes are described and their relationship to scrapie is discussed.

7. Failure to obtain any improvement in cases of scrapie and myopathy when treated with a number of nutritional, endocrine, and other therapeutic agents is recorded, with the exception of cases of P3 which often recover spontaneously.

8. The epidemiology and possible aetiology of these ovine myopathic diseases are considered. It is suggested that they may prove to be inherited, but subject to modification by environmental factors.

9. The organisation of flockmasters and shepherds into a voluntary Sheep Health Scheme for the purpose of investigating and attempting to control the disease is recorded.

10. The implications of these results on the present restrictions on the international trade in breeding sheep are considered.

11. The possible relationship of these ovine myopathies to human dermatomyositis (polymyositis) and muscular dystrophy is discussed briefly.

Acknowledgments.—I should like to thank the Nuffield Foundation, whose generous support made this work possible; my collaborators Dr. F. D. Bosanquet and Dr. P. M. Daniel for unstinted help; Dr. G. S. Dawes for his great interest and encouragement; the general veterinary surgeons and Veterinary Investigation Officers who have helped with some of the general problems arising in Health Scheme flocks; and finally the many flockmasters and shepherds, and the Suffolk Sheep Society, who have assisted so valiantly in keeping records and discussing freely many practical aspects of these diseases.

References

ANON. (1956). *Lancet.* **ii.** 767.
BESNOIT, —, & MOREL, C. (1898). *Rev. vét.* **23.** 397.
BLAXTER, K. L., & MCGILL, R. F. (1955). *Vet. Rev. Annot.* **1.** 91.
BOSANQUET, F. D., DANIEL, P. M., & PARRY, H. B. (1956a). *Lancet.* **ii.** 737.
———, ———, & ———. (1956b). *Ibid.* **ii.** 1355.
BROWNLEE, A. (1940). *Vet. J.* **96.** 254.
COTCHIN, E. (1948). *Ibid.* **104.** 102.
CUILLE, J., & CHELLE, P.-L. (1936). *C. R. Acad. Sci.* **203.** 1552.
———, & ———. (1938). *Ibid.* **206.** 1687.
GORDON, W. S. (1946). *Vet. Rec.* **58.** 516.
GREIG, J. R. (1940). *Trans. Highl. agric. Soc., Scot.* **52.** 71.
HOBMAIER, M. (1925). *Arch. wiss. prakt. Tierheilk.* **52.** 38.
HOLMAN, H. H., & PATTISON, I. H. (1943). *J. comp. Path.* **53.** 231.
M'FADYEAN, J. (1918). *Ibid.* **31.** 102.
M'GOWAN, J. P. (1914). "Investigation into a Disease of Sheep called 'Scrapie.'" Edinburgh: Blackwood.
MARR, T. G., SHARMAN, G. A. M., & BLAXTER, K. L. (1956). *Vet. Rec.* **68.** 408.
PARRY, H. B. (1949). *Ibid.* **61.** 23.
SEIFRIED, O. (1944). "Vitamine und Vitaminmangelkrankheiten bei Haustieren." Stuttgart: Ende. p. 111.
SHOLL, L. B. (1939). *J. Amer. vet. med. Assoc.* **95.** 108.
STOCKMAN, S. (1913). *J. comp. Path.* **26.** 317.
———. (1926). *Ibid.* **39.** 42.
VAWTER, L. R., & RECORDS, E. (1939). *J. Amer. vet med. Assoc.* **94.** 489.
WILLMAN, J. P., LOOSLI, J. K., ASDELL, S. A., MORRISON, F. B., & OLAFSON, P. (1946). *Cornell Vet.* **36.** 200.
WILSON, D. R. (1952). *Vet. Rec.* **64.** 468.
———, ANDERSON, R. D., & SMITH, W. (1950). *J. comp. Path.* **60.** 267.
ZIEGLER, M. (1929). In Joest, E. "Handbuch der speziellen pathologischen Anatomie der Haustiere." Vol. v. 383. Berlin: R. Schoetz.

MINISTRY OF AGRICULTURE REGIONAL CONTROLLERS

The Minister of Agriculture, Fisheries and Food, the Rt. Hon. D. Heathcoat Amory, M.P., has selected the following officers to become in due course the Ministry's regional controllers in the regions indicated in England:—

Northern Region, Mr. F. M. Kearns, M.C.; Lancs. and Yorks. Region, Mr. K. Harrison Jones; West Midland Region, Mr. W. J. B. Hopkinson, O.B.E.; East Midland Region, Mr. E. Ll. Harry, O.B.E.; Eastern Region, Mr. J. H. Perrin; S. Western Region, Mr. H. Pitchforth; S. Eastern Region, Mr. J. W. Pugsley.

It is expected that these officers will take up their new posts on April 1st next.

In reply to a question in the House of Commons on August 2nd, 1956, Mr. Heathcoat Amory said that he had decided to accept the recommendation of Sir Arton Wilson's Committee that he should appoint regional controllers. He added: "At present there are several organisations operating separately at regional level, due partly to the wide range of work involved and partly to the recent merger of the Agriculture and Food Departments. The proposals in the report will lead to greater uniformity and co-ordination; will secure proper supervision and increased efficiency of non-technical work and staff in the provinces; and will permit the devolution of many establishment, finance, and defence planning matters from Headquarters. The regional controllers will not intervene in the technical work of the main technical services or in the operational control of those services from Headquarters."

The list does not include an appointment in Wales where Mr. J. Morgan Jones, C.B.E., holds the appointment of Welsh Secretary.

Parenteral Coagulants in the Horse

BY

R. K. ARCHER

Equine Research Station, Animal Health Trust, Newmarket

Introduction

HAEMORRHAGE from various parts of the body often confronts the veterinary surgeon as a problem to be solved. Haemorrhage from large vessels on the surface of the body following trauma or during surgical interference is in a category by itself and can usually be adequately dealt with by mechanical means. On the other hand internal haemorrhages from injuries, following parturition, or from various obscure disturbances of blood pressure, heart's action, etc., present a problem which calls for the use of a parenterally-administered coagulating agent. Epistaxis of the race-horse, steeple-chaser, or hunter, may be cited as one instance of a clinical condition in which, if a reliable coagulant could be administered, it would have a useful application. It is, however, hardly necessary to emphasise that epistaxis is only the end result of some other disturbance, and may have several different causes. At present, treatment in established cases is usually possible though often unnecessary, but prevention of the haemorrhage is still largely an impossibility.

During recent years there have been a number of references in the literature to the veterinary clinical use of a coagulant consisting of oxalic acid and malonic acids in solution (Davies, 1939; Miller & Davies, 1939; Stapleton, 1942). Such preparations* are claimed to reduce, to a significant extent, the coagulation time of blood in normal animals. Steinberg & Brown (1939) reported that extracts of certain plants (*e.g.* beets, citrus fruits) or of liver or cord blood would, upon injection, " accelerate markedly the coagulation rate of the blood of rabbits." The presence of a dicarboxylic acid was established in each of these extracts. Martin (1940), using chicks and rabbits, demonstrated a decrease in clotting time about one hour after an intravenous dose of oxalic or malonic acid at a dosage level of 3 mg. per kg. Doses of 100 mg. per kg. increased the coagulation times.

Russell viper venom has long been used as a local haemostatic but is unsuitable for hypodermic injection. Klobusitzky (1954) postulated the existence of an enzyme, which he termed " haemocoagulase," in viper venom. This substance has been prepared in a form said to be free from toxins and proteins and is marketed under a trade name†. The product has been used in human clinical medicine in Austria and Switzerland (Bruck & Salem, 1954; Fleischhacker, 1954; Glaniger, 1955; Heiss, 1954), but no controlled clinical investigation has been published. There has been no report of veterinary use of this product.

* " Haemostem," Crookes Laboratories Ltd., London; " Koagmin," Austin Laboratories Ltd., Guelph, Canada; " Venagmin," Willows Francis Ltd., London.

† " Reptilase," Pentapharm, A. G., Basle.

An investigation of the use of oxalic and malonic acid preparations, and of " haemocoagulase," has been made at this Station using experimental ponies under carefully controlled circumstances. In view of the inevitable inaccuracy of the measurement of whole-blood clotting time, further work has been conducted by using recalcified equine plasma *in vitro* (Quick, 1942).

Methods

In vitro tests were performed upon recalcified equine plasma using a modification of the technique of Quick (1942). Blood was collected from four normal horses into 0.1 *M.* trisodium citrate, using one part of anticoagulant to nine parts of blood. This was centrifuged for 10 minutes at 3,000 r.p.m. and the plasma was pipetted off. A solution of calcium chloride, 0.025 *M.*, was prepared. Serial dilutions were made in normal saline of the parenteral coagulant under investigation. The various reagents were placed in tubes in a water bath accurately maintained at 37.5° C. Tests were made by adding solutions with " blow-through " pipettes to standard test-tubes 3 inches by $\frac{3}{8}$ inch in the water bath in a definite order, as follows:—

(1) 0.1 ml. equine plasma.
(2) 0.1 ml. coagulant, or, for controls, saline.
(3) 0.1 ml. 0.25 *M.* calcium chloride.

At the moment of addition of the calcium solution an assistant started a stop watch. The tube was inverted every 10 seconds until a complete clot was formed, when the time was noted. This time was recorded.

A further *in vitro* system was set up which was precisely similar to the first except that lipid co-factor was added to the system. This can be derived from brain by extraction with chloroform or it can be satisfactorily simulated by ordinary milk cream, 1 part, in 10 parts of normal saline. The second series of tests was set up by adding solutions to the standard test-tubes in the following order:—

(1) 0.1 ml. equine plasma.
(2) 0.1 ml. lipid co-factor in saline.
(3) 0.1 ml. coagulant, or, for controls, saline.
(4) 0.1 ml. 0.025 *M.* calcium chloride.

The chief effect of lipid co-factor is markedly to accelerate the coagulation time.

In vivo. Two ponies, approximating to the same weight (356 and 300 lb., respectively), were maintained together in the same box. One pony received an intravenous dose of the coagulant under investigation, while the second pony was maintained as a control. Clotting times of whole-blood were determined upon both ponies according to the method of Lee & White (1913) immediately prior to the injec-

tion of the coagulant and at varying intervals thereafter up to 24 hours.

There are wide variations in the normal clotting time of equine blood determined by the Lee & White method and much of this is referable to time of day, feeding (fat content of the blood), state of alarm (activity of suprarenal gland), etc., etc. In an attempt to minimise the misleading effect of these variations, the experiment was so designed that controls from one pony to the other would show changes attributable to environment or feeding; whereas, controls upon the same pony at a later or an earlier time would reveal individual idiosyncrasies.

The ponies were used alternatively as experimental and control animals and at least 48 hours was left between the end of one experiment upon them and the commencement of another.

Results

The *in vitro* results using recalcified plasma are given in Fig. 1 and Table I. It will at once be seen that at no time has the addition of an oxalic and malonic acid preparation accelerated coagulation beyond the limit of controls, regardless of the dilution of the product. Indeed, if this dilution were less than 1:10 then coagulation failed to occur for a

TABLE I
RECALCIFIED PLASMA SYSTEM *In vitro*

Reciprocal of dilution of coagulant	Range of coagulation times. Decrease (−) or increase (+) over individual controls							
	"Reptilase"		"Haemostem"		"Koagmin"		"Venagmin"	
1	−3′ 00″	−2′ 30″	+60′	+60′	+60′	+60′	+60′	+60′
2	−2′ 30″	−1′ 50″	+60′	+60′	+60′	+60′	+60′	+60′
4	−1′ 05″	−0′ 50″			+3′ 00″	+8′ 00″		
10					−0′ 30″	+0′ 20″		
20					−0′ 40″	+0′ 10″		
50	−0′ 20″	−0′ 10″	−0′ 05″	+0′ 05″	−0′ 10″	+0′ 40″	−0′ 15″	+0′ 30″
100	−0′ 05″	+0′ 05″	−0′ 05″	+0′ 05″	−0′ 10″	+0′ 15″	−0′ 10″	+0′ 35″
1,000	−0′ 05″	+0′ 10″	−0′ 05″	+0′ 06″	−0′ 10″	+0′ 30″	−0′ 05″	+0′ 50″

Note.—Each test was performed in quadruplicate with an individual control in every case. Four plasmas were used. Average control coagulation time, 5′ 30″.

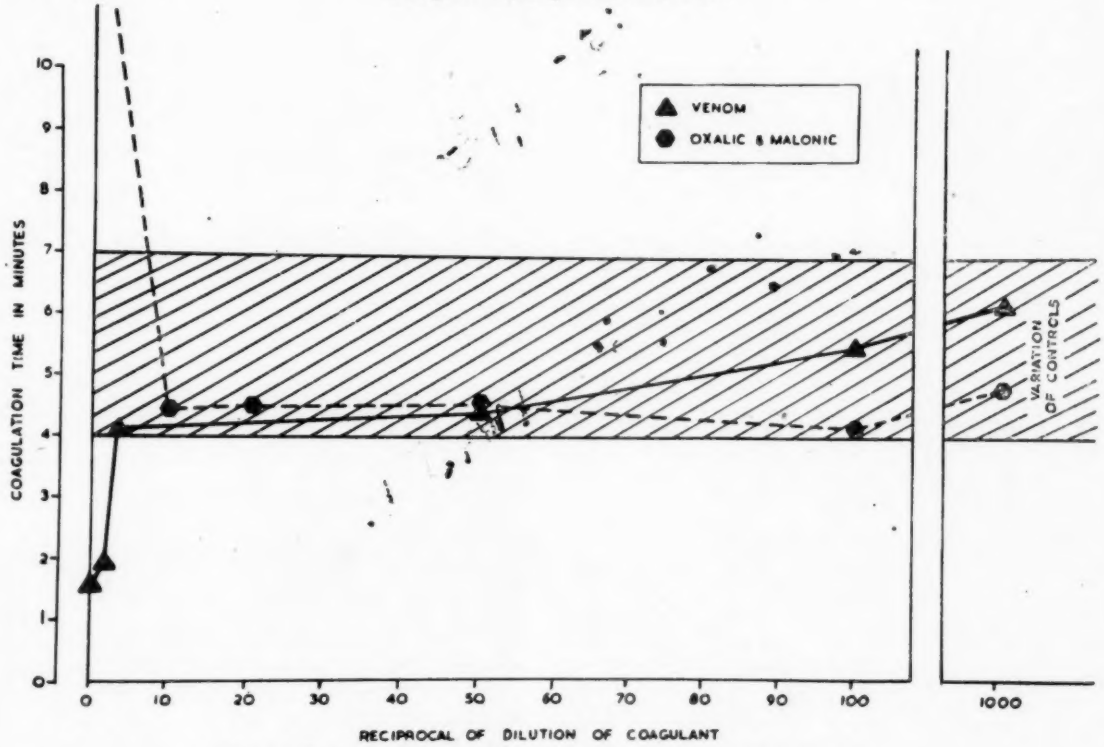

FIG. 1.—COAGULATION TIME OF AN *in vitro* RECALCIFIED PLASMA SYSTEM.
The three preparations of oxalic and malonic acids investigated have been plotted as one curve since no material difference was apparent between them.
The points plotted are the average of all test times at the various dilutions of coagulant investigated. All controls were between the limits of 5′ 30″ ± 1′ 30″.

long period. It was possible to see a white precipitate formed in the tube at low dilution of the reagent, presumably of calcium oxalate.

The effect of " Reptilase " was strikingly different from that of oxalic and malonic acids in that there was a highly significant decrease in the coagulation time. However, this increase was only noticeable at a very high concentration of the coagulant (more than 1:4), such a concentration being much higher than it would be possible to achieve in the intact animal.

Repetitions of these tests using lipid co-factor are illustrated in Fig. 2 and Table II. It will be seen that any possible potentiation of the coagulants investigated by the presence of lipid can be entirely discounted. The dilution at which " Reptilase " produced a significant decrease in coagulation time was unchanged (more than 1:4); while the effect of

TABLE II

RECALCIFIED PLASMA SYSTEM *in vitro* WITH LIPID CO-FACTOR

Reciprocal of dilution of coagulant	Range of coagulation times. Decrease (−) or increase (+) over individual controls							
	" Reptilase "		" Haemostem "		" Koagmin "		" Venagmin "	
1	−65″	−55″	+60′	+60′	+60′	+60′	+60′	+60′
2	−50″	−45″			+360″	+480″		
4	−10″	−05″			+ 20″	+ 50″		
10					+ 10″	+ 40″		
50	0	+01″			+ 05″	+ 07″		
100	+01″	+10″	−01″	+13″	− 02″	+ 08″	+03″	+15″
1,000	+03″	+07″	−03″	+12″	− 02″	+ 12″	+04″	+25″

Note.—Each test was performed in quadruplicate with an individual control in every case. Four plasmas were used. Average control coagulation time, 112″.

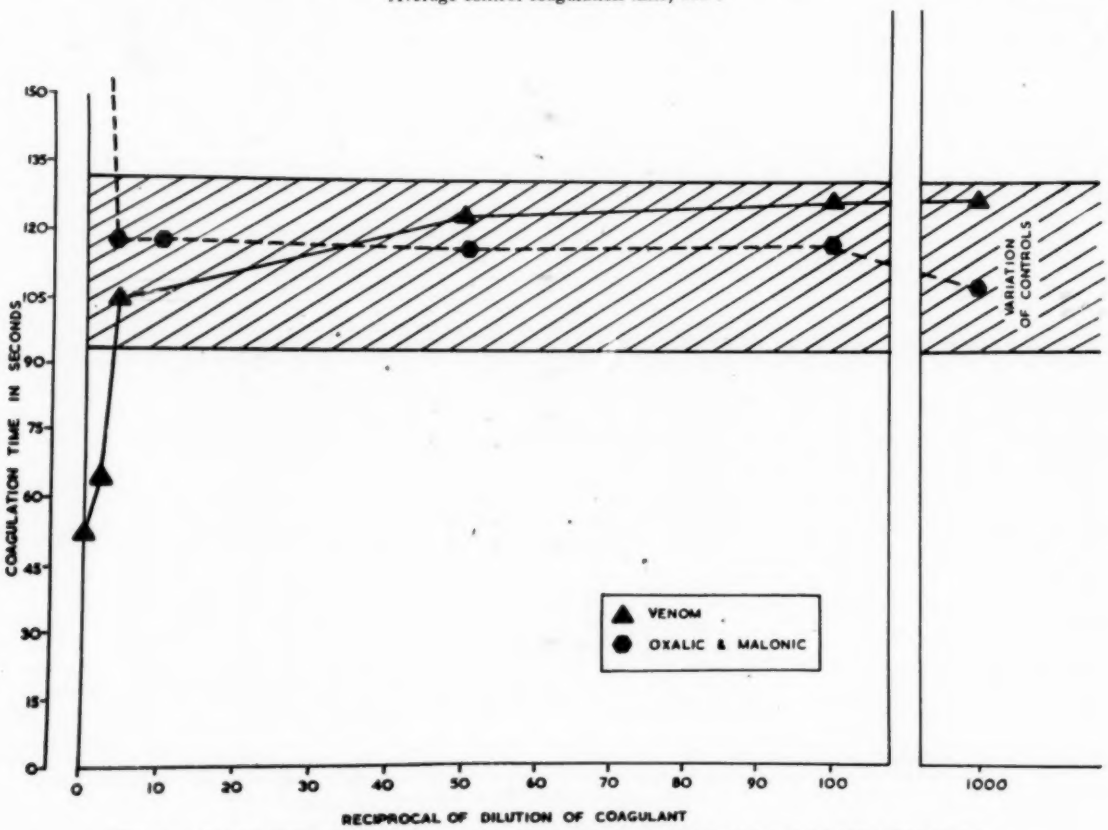

FIG. 2.—COAGULATION TIMES OF AN *in vitro* RECALCIFIED PLASMA SYSTEM WITH LIPID CO-FACTOR.

The three preparations of oxalic and malonic acids investigated have been plotted as one curve since no material difference was apparent between them.

The points plotted are the average of all test times at the various dilutions of coagulant investigated. All controls were between the limits of 112″ ± 18″.

oxalic and malonic acids was never to accelerate coagulation.

The curves drawn in Figs. 1 and 2 illustrating the *in vitro* effect of coagulants are, in each case, the average of four replicate tests using four different plasmas. Every test was accompanied by its own control and every control time fell within the limits drawn upon the figures. No differences were apparent between the three preparations of oxalic and malonic acids investigated and the results are plotted together.

Fig. 3 and Table III show the *in vivo* results obtained with the four products investigated. Again, no material difference was apparent between the various preparations of oxalic and malonic acids. None of the parenteral coagulants investigated produced coagulation times outside the limits of normality even when used at double the recommended therapeutic doses. There is a movement in the limits of control variations with time after repeated bleeding in the horse which is represented in Fig. 3. This is probably due to a combination of the effect of stimulation of the animals associated with handling and also the increased lipid concentration in peripheral blood following a meal. Each test was commenced at 10.00 a.m. and the animals were fed their oat ration at 9.00 a.m. and hay at 10.30 a.m. identically throughout the whole period of observation.

Discussion

Oxalic and malonic acids in solution as supplied by three pharmaceutical firms failed to decrease the coagulation time of peripheral blood in horses either at the recommended dose or at about double that dose (*i.e.* 1.2 mg. per kg. intravenously to a pony which is to be compared with the recommended dose of 0.5 mg. per kg. intravenously for a Thoroughbred). The *in vitro* investigations did not show any evidence that the agents could reduce the coagulation time of recalcified equine plasma; indeed they increased this time very considerably unless diluted at least 1:10. In the report by Steinberg & Brown (1939), in which a decrease in clotting time of rabbit blood is associated with injections of various extracts, no *in vitro* work was performed. Martin (1940) reported *in vitro* work using rabbit's blood in which he recorded a reduction in clotting time of 5 to 60 per cent. of the normal after addition of 0.2 ml. of 0.001 *M*. sodium oxalate. This corresponds approximately to a dilution of 1:10

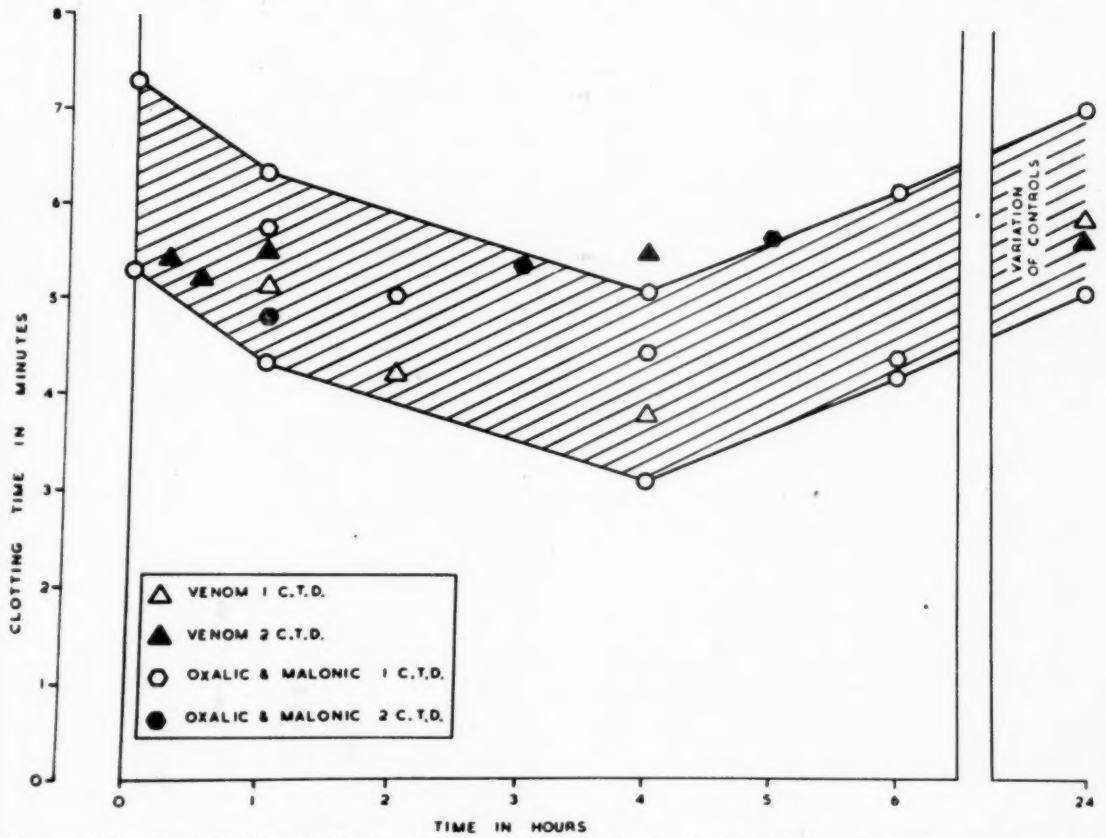

FIG 3.—CLOTTING TIMES (LEE & WHITE METHOD) OF EQUINE BLOOD *in vivo* AFTER INTRAVENOUS INJECTION OF COAGULANTS.

The average control clotting times fell within the limits drawn. "Hollow" plots represent one calculated therapeutic dose (1 C.T.D.); "solid" plots represent two calculated therapeutic doses (2 C.T.D.).

TABLE III
In vivo EXPERIMENTS. CLOTTING TIMES (Method of Lee & White)

Date of experiment	1st	1st	4th	4th	7th	7th	10th	10th	
Animal	Pony I	Pony II	Pony I	Pony II	Pony II	Pony I	Pony II	Pony I	
What injected I/V.	" Reptilase " 2 ml.	Control	" Venagmin " 20 ml. (=1·24 mg./kg.)	Control	" Reptilase " 4 ml.	Control	" Koagmin " 10 ml. (=0·74 mg./kg.)	Control	Approximate control average
0 hour	6′ 43″	6′ 37″	5′ 43″	7′ 50″	5′ 30″	5′ 29″	6′ 24″	5′ 36″	6′ 20″
15′	—	—	—	—	5′ 26″	3′ 58″	—	—	—
30′	—	—	—	—	5′ 14″	3′ 25″	—	—	—
1 hour	5′ 10″	4′ 50″	4′ 50″	6′ 30″	5′ 32″	4′ 23″	5′ 40″	6′ 04″	5′ 20″
2 hours	4′ 15″	4′ 58″	—	—	—	—	5′ 05″	7′ 03″	—
3 „	—	—	5′ 25″	5′ 55″	—	—	—	—	—
4 „	3′ 50″	3′ 35″	—	—	5′ 52″	2′ 55″	4′ 30″	5′ 34″	4′ 10″
5 „	—	—	5′ 43″	6′ 05″	—	—	—	—	—
6 „	—	—	—	—	—	—	4′ 20″	5′ 10″	5′ 10″
24 „	5′ 55″	7′ 22″	—	—	5′ 44″	4′ 55″	—	—	6′ 10″

Pony I weighed 356 lb. Pony II weighed 300 lb. Both ponies were uncastrated yearlings.

of the commercially prepared oxalic and malonic acid preparations and is far higher than the recommended therapeutic dosage for equidae (only 0.5 mg. per kg.). Details of the *in vitro* system used in Martin's work are not given.

It is notoriously difficult to draw conclusions from clinical trials of such products because of the extreme difficulty of adequate control. It is hardly possible for a veterinary surgeon deliberately to withhold an agent he believes to be effective in a series of cases merely for scientific observation and, even if that were to be done, it is impossible to achieve exact replicates of, for example, bleeding states. If it be noted that a series of subjects bled less than previous experience would lead the observer to expect, as a result of an injection of any agent, it is not possible to exclude the effect of a series of unknown additional factors. Should a surgical case be involved, the haemostasis may have been achieved more thoroughly, perhaps because of better technique. If accidental damage is concerned, then the effect of tissue fluid on the one hand, and of the vessels broken by the injury on the other hand, must necessarily remain unknown. In the series of clinical papers published upon the use of oxalic and malonic acids to reduce the clotting time of blood, there has been little or no attempt to achieve any controlled observation. It seems certain, from the results reported here, that oxalic and malonic acid preparations can have no place in the therapeutic armamentarium of the equine practitioner.

The case of " Reptilase " is somewhat more complex, although the published clinical material (referring entirely to man) is without adequate control. The *in vitro* work described here is suggestive that this agent might have an effect upon the equine coagulation system if present in sufficient quantity. But to achieve a concentration at the site of bleeding approaching 1:4 is not practicable by intravenous or hypodermic injection. *In vivo* experiments using about double the recommended therapeutic dose failed to show any significant decrease in clotting time.

Even if any of the agents investigated had produced a significant reduction in clotting time in the intact normal horse, it would not have followed that an increase in clotting efficiency of any practical value would ensue. Had clotting time been reduced by half, let us say from seven to three-and-a-half minutes, that would hardly be of appreciable value in capillary oozing. Clearly no effect upon frankly bleeding larger vessels would occur. It should be emphasised that the whole of this work is based upon the equine subject, and horses are known to have a clotting system differing in a material way from that of man (Bell, Tomlin & Archer, 1955).

Conclusions

1. Oxalic and malonic acids 1 per cent. a.a. have no effect in reducing the clotting time of the blood of the normal horse at concentrations up to 1.2 mg. per kg. (double the recommended dose).

2. Haemocoagulase (" Reptilase "), a product made from the venom of certain snakes, has no effect in reducing the clotting time of the blood of the normal horse at a dosage of as much as double the recommended rate.

Acknowledgments.—I am grateful to Mr. William C. Miller, Director of the Equine Research Station, for his help and encouragement with this work. I am obliged to Mr. R. E. Ablett of A. & G. Nicholas Ltd., Slough, for supplies of " Reptilase." I have had able technical assistance from Miss Diana Quinn of the Equine Research Station.

References

BELL, W. N., TOMLIN, S. C., & ARCHER, R. K. (1955). *J. Comp. Path.* **65.** 255.
BRUCK, H., & SALEM, G. (1954). *Wien. Klin. Wschr.* **66.** 395.
DAVIES, A. A. (1939). *J. Vet. Med.* **95.** 510.
FLEISCHHACKER, H. (1954). *Wien. Med. Wschr.* **104.** 171.
GLANIGER, J. (1955). *Mon. Fur Ohren. Und Lar.-Rhin.* **89.** 63.
HEISS, H. (1954). *Wien. Med. Wschr.* **104.** 833.
KLOBUSITZKY, D. (1954). *Klin. Med.* **9.** 310.
LEE, R. I., & WHITE, P. D. (1913). *Amer. J. Med. Sci.* **145.** 495.
MARTIN, G. J. (1940). *Amer. J. Physiol.* **130.** 574.
MILLER, L. J., & DAVIES, A. A. (1939). *Vet. Med.* **34.** 544.
QUICK, A. J. (1942). "The Haemorrhagic Diseases and the Physiology of Haemostasis." Thomas, Springfield, U.S.A.
STAPLETON, S. F. (1942). *Fort Dodge Biochem. Rev.* **13.** 24.
STEINBERG, A., & BROWN, W. R. (1939). *Amer. J. Physiol.* **126.** 638.

Legal Pitfalls of Veterinary Practice

BY
CHARLES STEVENS

IV.—NEGLIGENCE

OF all civil actions with which a veterinary surgeon finds himself threatened, that on negligence is the most frequent, and it is, therefore, advisable to say something about this particular tort.

Negligence has been described by a great English common lawyer in the following terms:—

> "Negligence as a tort is the breach of a legal duty to take care which results in damage, undesired by the defendant, to the plaintiff."

The responsibilities and the degree of care in practice required from the veterinary surgeon were discussed in the first article of this series, and it is not intended to expand on that aspect further.

It should be noted, however, that although it is only breach of a legal duty which gives rise to an action, there is no doubt that in the relationship between the veterinary surgeon and his client a legal duty of care always exists. No veterinary surgeon would be deliberately careless, but in the pressure of practice a slip could occur, and it is immaterial whether the harm resulting is desired or envisaged. All that it is necessary for the plaintiff to prove is that harm to his animal was the result of treatment or lack of treatment from the veterinary surgeon, of a standard less than normal.

Examples of negligent actions which have given rise to claims in the past are the following:—

Five pigs were castrated and the first three died of tetanus. It was alleged that the veterinary surgeon was negligent in not taking preventive steps after occurrence of the first death, and a settlement had to be made. In another instance, concerning a pedigree Ayrshire, it was shown by expert evidence that had pregnancy been diagnosed—which the veterinary surgeon should normally have done—expression of Corpus would not have been necessary. Here again a cash settlement had to be made to avoid an action that would surely have gone against the veterinary surgeon. A third instance was where swine fever had been brought to a herd by a sow, and this sow had been seen by a veterinary surgeon who stated its death was due to gangrene of the udder. A *post mortem*, which would have been reasonable in the circumstances, would have revealed swine fever—as in fact the carcase did, on exhumation at a later date. It was held that the veterinary surgeon was negligent in not taking the precautions his professional training would have shown him to be necessary. This claim was settled only by a considerable payment. In all three cases the veterinary surgeons were members of the National Veterinary Benevolent and Mutual Defence Society and the claims were met in full by that Society.

Normally, negligent mistatements are not actionable, but they do become actionable when there is a contractual relationship between the parties concerned. There is always at law a contractual relationship between the veterinary surgeon and his client, and therefore a veterinary surgeon must avoid negligence not only in his actions but also in his statements when dealing with clients.

It was pointed out in the first article in this series that the degree of care and attention required from the veterinary surgeon is only that of the average or reasonable veterinary surgeon, and two cases defended by the Defence Society where claims were not successful and were abandoned illustrate this point.

The first was a claim for £200 for a bull which died under chloroform. Death under an anaesthetic is a risk that may arise at any time, and as it was shown that the veterinary surgeon used accepted methods of practice and was in no way negligent, the claim could not be substantiated and was withdrawn.

The other claim was for undue losses in anaemic piglets, where the stockowner said the losses had occurred because the veterinary surgeon had prescribed anaemia capsules instead of iron-copper mixture. This claim, also, could not be substantiated because the veterinary surgeon was treating according to the best of his ability and it could not be said that he was negligent or had tried out some unknown method which gave rise to the losses.

When a stockowner places his animal in the care of a veterinary surgeon the responsibility in the main passes from the stockowner to the veterinary surgeon, and the unqualified person is not entitled to dictate methods of treatment or the drugs to be prescribed, nor will a cause for action lie if the result of the treatment is not as successful as the stockowner thought possible if he had taken a hand in it himself.

This last statement raises the question of contributory negligence. It is possible for the stockowner or his employees to be negligent in addition to the veterinary surgeon. For example a veterinary surgeon may see the animals, give treatment, and prescribe a course of action that must be followed until his next visit. It may be that the stockowner or his employees ignore the instructions of the veterinary surgeon as a result of which damage occurs. If the negligence of the stockowner is solely responsible for the loss, no action at all will lie against the veterinary surgeon; but if the veterinary surgeon also was negligent and the loss occurred through the negligence of both, the action will still lie but damages will be apportioned. At one time contributory negligence by the plaintiff barred an action altogether, but this is no longer so, and the courts will entertain an action and themselves decide how the blame should be apportioned and the damages divided.

Lastly, one should point out that no matter how careful a veterinary surgeon may be in his actions and

(*Continued at the foot of col.* 1, *page* 62)

News and Comment

SUPPLEMENTARY SUPPLIES OF PETROL

The B.V.A. has taken up with the Ministry of Transport and Civil Aviation the question of supplementary supplies of petrol for members of the profession who use dual-purpose or similar vehicles carrying a goods licence.

Arrangements have been made for the procedure to be the same as that agreed with the Ministry of Fuel and Power for dealing with applications for supplementary coupons in respect of private cars.

Thus applications for supplementary petrol for goods vehicles should continue to be made, in the first place, to the appropriate Regional Transport Commissioner. Any member who may meet with difficulty is advised to contact his B.V.A. Liaison Officer for help. A list of such officers was published in THE VETERINARY RECORD of December 29th, 1956.

RABBIT CLEARANCE

Although grants of 50 per cent. are available for clearing rabbit-infested scrub, one of the difficulties which has been met in the anti-rabbit campaign is the clearance of rabbit harbourage on land which forms no part of an agricultural unit. Therefore, a higher rate of grant of up to 75 per cent. of the cost is now being offered as an incentive to owners or occupiers of non-agricultural land who would derive no economic benefit from clearance. Where clearance is not practicable, these grants may be given for fencing-in rabbit-infested areas.

Owners and occupiers who receive these grants will be required to give an undertaking either to ensure that the scrub does not become re-established or to maintain the fencing in good repair. The cost of gassing powder, which is one of the most humane and effective methods for destroying rabbits, is subsidised, and rabbits on commons and derelict lands are dealt with at public expense.

Further details of the grants can be obtained from the Ministry's county offices which can also arrange for advice to be given on the best method of dealing with a rabbit-infested area.

BADMINTON HORSE TRIALS, 1957

In spite of the petrol situation it has been decided that these Horse Trials, which have now become one of the main events in the Equestrian Calendar, will be held, as originally planned, at Badminton, Gloucestershire, by kind permission of His Grace The Duke of Beaufort, on April 25th, 26th, and 27th.

As usual, reserved seats in the stands for the Dressage and Jumping phases on the first and last days will be available.

UNITED KINGDOM AGRICULTURAL AND FOOD REPRESENTATIVE IN DENMARK AND THE NETHERLANDS

Mr. M. Whalley Taylor, M.B.E., M.A., Chief Poultry Advisory Officer for the West Midland Province of the National Agricultural Advisory Service, has been appointed Agricultural and Food Attaché to H.M. Ambassadors at Copenhagen and the Hague. He will also hold a watching brief on technical agricultural developments and food trade trends in Western Germany. He is due to arrive in Copenhagen on or about January 28th.

IMPORTED MEAT

Upton-on-Severn N.F.U. are concerned at the apparent regularity with which outbreaks of foot-and-mouth disease occur, and want greater measures taken to prevent the importation of infected meat. The following resolution, to be forwarded to the County Executive, has been approved: "Since a number of outbreaks of foot-and-mouth disease have been traced to imported meat, the Branch feels that greater measures should be taken to prevent infected meat and offal entering the country."

UNIVERSITY NEWS

Bristol

Final Examination for the Degree of B.V.Sc. December, 1956

Pass

Al-Hammawi, M. H.; Mason. R. S.; Ryland. D. N.; Thomas, E. H.

R.C.V.S. OBITUARY

We record with regret the deaths of the following members of the profession:—

CROWE, John Martin, Monasteroris, Edenderry, Co. Offaly, Eire, Capt. late R.A.V.C. Graduated Dublin, July 24th, 1911. Died January 3rd, 1957, aged 70 years.

ROBINSON, Henry W., 2, Clover Hill Park, Massey Avenue, Belfast. Graduated London, December 16th, 1904. Died January 9th, 1957, aged 76 years.

COMING EVENTS

January

24th (Thurs.). Annual Dinner and Dance of the V.V.B.F. (Northern Division) in the Northern Hotel, Aberdeen, 7.30 p.m.

25th (Fri.). Annual General Meeting of the Yorkshire Division at the Hotel Metropole, Leeds, 2.30 p.m.

Legal Pitfalls of Veterinary Practice—*(concluded)*.

statements, there is always the type of person who will endeavour to recoup himself for inevitable losses by threatening to bring a legal action. The veterinary surgeon who has not been negligent need have little fear of such threats, as it is rarely, if ever, that claims of this nature would be pushed as far as a court action.

30th (Wed.). General Meeting of the Lancashire Veterinary Association at the Midland Hotel, Manchester, 2.30 p.m.

Ordinary General Meeting of the Sussex Veterinary Society at the Old Ship Hotel, Brighton, 2.30 p.m.

February

1st (Fri.). Annual General Meeting of the Mid-West Veterinary Association at the Berkeley Café, 2.30 p.m.

Annual General Meeting of the Northern Ireland Veterinary Association at the Union Hotel, Donegall Square, Belfast, 6.30 p.m.

6th (Wed.). General Meeting of the Ayrshire Veterinary Association in the Hotel Dalblair, Ayr, 2.30 p.m.

7th (Thurs.). General Meeting of the Central Veterinary Society at the Royal Veterinary College, Camden Town, London, N.W.1, 6 p.m.

13th (Wed.). General Meeting of the Society of Practising Veterinary Surgeons at the Royal Veterinary College, Camden Town, N.W.1, 2.30 p.m.

19th (Tues.). 81st Annual General Meeting of the Eastern Counties Veterinary Society at the Bell Hotel, Norwich, 11.15 a.m.

21st (Thurs.). Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 8.30 p.m.

BRITISH VETERINARY ASSOCIATION
QUARTERLY MEETINGS

Wednesday, January 23rd, 1957, at 7, Mansfield Street, W.1.

10.00 a.m. Parliamentary and Public Relations Committee.
11.30 a.m. Veterinary State Medicine Committee.
2.00 p.m. Small-Animals Committee.

Thursday, January 24th, 1957, at 7, Mansfield Street, W.1.

10.00 a.m. Farm Live-stock Committee.
11.30 a.m. Home Appointments Committee.
12.30 p.m. Finance Sub-committee.
2.30 p.m. General Purposes and Finance Committee.

Friday, January 25th, 1957, Connaught Rooms, Great Queen Street, W.C.2.

10.30 a.m. Extraordinary General Meeting followed by Council Meeting.

FOOT-AND-MOUTH DISEASE

The area described in the schedule below is declared to be an Infected Area for the purpose of preventing the spread of foot-and-mouth disease:—

The County Borough of Southend-on-Sea.

IN THE COUNTY OF ESSEX

The Borough of Maldon.

The Petty Sessional Divisions of Dengie and Maldon and Rochford.

In the Petty Sessional Division of Witham: The parishes of Hatfield Peverel, Wickham Bishops, Ulting, Great Totham, Little Totham, Goldhanger and Tollenshunt Major.

In the Petty Sessional Division of Chelmsford: The parishes of Boreham, Springfield, Little Baddow, Great Baddow, Sandon, Danbury, West Hanningfield, East Hanningfield, Woodham Ferrers and Rettendon and so much of the parishes of South Hanningfield and Runwell as lies within the Petty Sessional Division.

In the Petty Sessional Division of Billericay: So much of the parishes of South Hanningfield and Runwell as lies within the Petty Sessional Division and so much of the parish of Billericay as lies to the east of the road (B.1007) running from Stock to Great Malgraves excluding the said road.

The Foot-and-Mouth Disease Infected Area around Brede, East Sussex, will be released from restrictions on January 22nd:—

IN THE COUNTY OF EAST SUSSEX

The Borough of Rye.

In the Petty Sessional Division of Rye: The parishes of Northiam, Beckley, Peasmarsh, Rye Foreign, Udimore, Icklesham, Brede, Guestling, Pett and Fairlight and so much of the parish of Iden as lies to the west of the road (B.2082) running from Wittersham to Playden excluding the said road.

In the Petty Sessional Division of Battle: The parishes of Ewhurst, Sedlescombe, Westfield and Ore.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ches. Hill Top Farm, Tushingham (Jan. 8).

Cumberland. Whitrigg Grange, Kirkbridge, Carlisle (Jan. 11).

Dorset. Manor Farm, Poxwell, Dorchester (Jan. 4).

Dunbarton. Netherwood Farm, Castle Cary (Jan. 7).

Hants. Garston Farm, E. Meon, Petersfield (Jan. 12).

Notts. Top Farm, Stoke Bardolph (Jan. 7).

Salop. Cross Green Farm, Berwick, Shrewsbury (Jan. 4); Rodington House Farm, Rodington, Shrewsbury (Jan. 11).

Staffs. Brough Hall, Gnosall (Jan. 7).

Sussex. Woodside Farm, Colgate, Horsham (Jan. 5).

Fowl Pest

Essex. 35, Nipsells Chase, Mayland, Chelmsford (Jan. 4); Aurora, Nipsells Chase, Mayland, Chelmsford (Jan 1); 3, Horkesley Road, Boxted, Colchester (Jan. 8); Heathcote, Langham Road, Boxted; Southdown, Langham Road, Boxted; White Lodge, Sandon, Chelmsford (Jan. 11); Steeple Hall, Steeple, Southminster; Orchard Leigh, Langham Road, Boxted (Jan. 12).

Herts. 117, Mullway, Letchworth (Jan. 6).

Lancs. Earnsdale, Grange Lane, Hutton (Jan. 4); Willow Farm, Black Leach Lane, Catforth; Park Lane, Forton; St. Michaels Cottage, Tag Lane, Ingol (Jan. 5); Marl Cottage, Marl Cop, Bretherton; 1, Edens Cottages, Woodplumpton (Jan. 7); Lower Holme, Slack Farm, Deepdale; The Allotments, Stoneygate Lane, Ribchester; Higher Morris Farm, Clayton-le-Wood, Chorley; Dansons Farm, Staynall, Hambleton (Jan. 8); Higher Oakenhead Farm, Whitechapel, Goosnargh; Nook Farm, Bretherton; Crosskeys Inn, Whitechapel, Goosnargh (Jan. 9); Moss House Farm, Stalmine, Fleetwood; Park Lane Poultry Farm, Forton (Jan. 10); Thatcher House Farm, St. Michaels; Ribchester Hospital, St. Michaels; 4, St. Heliers Place, Barton; Lunds Lane Farm, Much Hoole; Olivers Farm,

Eave, Woodplumpton; Parker's premises, Tag Lane, Ingol, Preston (Jan. 11); Mossley Farm, Eaves, Woodplumpton (Jan. 12); Little Carrside Farm, Warton, Preston (Jan 13).

Middx. Nazareth House, East End Road, Finchley, N.W.2 (Jan. 4).

Norfolk. Bridge Farm, Northrepps, Cromer (Jan. 4).

Yorks. Blackmoor Farm, Shadwell, Leeds (Jan. 7); Plot 19, Field End Road, Halton, Leeds (Jan. 9); Pilmoor Hall, Helperby (Jan. 11).

Swine Fever

Glamorgan. Piggeries, Cwrt-yr-Ala Road, Cyntwell Ely, Cardiff (Jan. 8); Fair Oak, New Cardiff Road, Mountain Ash (Jan. 10); Ynysygwas Farm, Cmavon, Port Talbot (Jan. 14).

Middx. Piggery, Rectory Farm, Tentelow Lane, Norwood Green, Southall (Jan. 9).

Northants. Luffield Abbey Farm, Silverstone, Towcester (Jan. 14).

Salop. New Foundland, Hughley, Shrewsbury (Jan. 14).

Staffs. 69, Holding, Watling Street, Weeford, Lichfield (Jan. 9); Sherrill Hall, Drayton Bassett, Tamworth; Hanbury Park Farm, Hanbury, Burton-on-Trent (Jan. 14).

Suffolk. Moss Farm, Worlingworth, Woodbridge (Jan. 10).

Warwicks. Common Farm, Ansley, Nuneaton (Jan. 10).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Content of "The Veterinary Record"

Sir,—I have been intrigued by the correspondence on the "Content of THE VETERINARY RECORD" initiated by my friend and near neighbour, Sidney Nye.

I realise that news is slow in percolating to some of the more underdeveloped areas of this county, but surely Mr. Nye must realise that no modern surgery is complete without a "Radio-active Isotope" or two about the place and that in more up-to-date areas of the county of Essex such articles as "The Trimethyl-amino-benzine requirements of the one-humped Caribou of Southern Tibet" are avidly read, while no meal-time passes without a lively discussion on lighter topics such as "Hexadecamethylene-bis-isoquinolinium chloride" to mention a few!!

Shame on you, Sidney, that you should wish THE RECORD to descend to such mundane topics as "Fractured Patellae in Greyhounds" Why! You will be expecting them to publish articles on the "Application of Red-hot Irons or Concentrated Acids to Horses' Legs" next.

For our more backward readers, Mr. Editor, may I suggest that an approach be made to either B.B.C. Television or I.T.V. with a view to having the more serious scientific articles reproduced in "serial form." Could we not visualise "The B.V.A. Show," including "Noddy among the Isotopes"; "Armed Conflict in the Alimentary Canal" (Commentary by Dr. Williams-Smith and Mr. Crabb); or what about "A Tetracycline Holiday with Bacillus Coli" or "This is your S.W.I.F.E." (sponsored by the Animal Health Division)?

Anything we can do, Sir, to bring enlightenment to the backward areas must be done at once—the time is Nye!!!

Whilst reading this week's RECORD my eyes fell on the "obituary type" notice announcing the appointment of a Public Relations Officer. Is it safe, I wonder, to offer such dazzling prizes as £1,250 a year to the successful applicant? Gad! How he will live! What a life of debauchery and gaiety. Entertaining the "Minister" at espresso bars, with long, exciting journeys by bus and bicycle.

For heaven's sake let's face facts! If we are to have a P.R.O. then let us be prepared to offer a salary which will attract a man of experience and ability. £1,250 a year! It is almost impossible to get a freshly qualified assistant for that money—and then you have to supply a car and a house!!

When I read that advertisement, Mr. Editor, it made me realise where Mr. Spike Milligan must get some of his material!

Yours faithfully,
ROGER MASSEY.

Mill House,
Stock,
Essex.

January 13th, 1957.

Acid Firing

Sir,—Acid firing, which was quite common 70 odd years ago, seems to be coming into its own again. More owners are enquiring about it and more are having it done in preference to the hot iron. I agree with Brigadier Peatt and Mr. Beckett that the results are just as good, with much less pain and discomfort to the horse.

I was first introduced to acid firing by a farrier veterinarian in my home town. When I was at school I used to spend a lot of my time with him. He used corks stuck on the ends of horseshoe nails, and he took the shine off the corks by rubbing them with a file. He always had the leg washed and thoroughly cleaned beforehand.

As regards after treatment, for the first three or four weeks, I insist on the horse being kept in a loose-box, and led out by hand once or twice a day. For the next three or four weeks he is allowed into a paddock or straw-yard, but no galloping is permitted. Following this, I allow a month's quiet road work at the walk (the patient being led beside another horse if possible), and then later I have the saddle put on and the horse is encouraged gradually to trot, to canter, and then to gallop.

I have tried Brigadier Peatt's method, but I have had difficulty in keeping the sleek sticking to the leg. It is not necessary to use sleek, but no doubt it looks more professional.

Yours faithfully,
J. F. RANKIN.

"Beverley,"
3, Alcombe Road,
Minehead.

January 12th, 1957.